



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

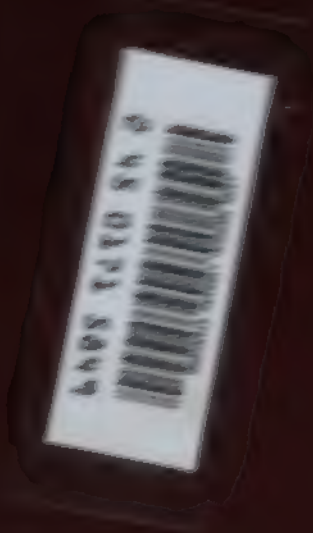
Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>



LANE

MEDICAL



LIBRARY

**THE BARKAN LIBRARY OF
OPHTHALMOLOGY AND OTOTOLOGY**

DISEASES OF THE EYE AND DISORDERS OF SPEECH IN CHILDHOOD

BY

PROF. DR. OSKAR EVERBUSCH
MUNICH

DR. MAX NADOLECZNY
MUNICH

TRANSLATED BY
LUCIEN HOWE, M.D., Sc.D.,
Fellow of the Royal Society of Medicine
LEE MASTEN FRANCIS,
Ph.B., M.D., F.A.C.S.
Buffalo, N. Y.

TRANSLATED BY
CLINTON P. McCORD, M.D.,
Health Director, Board of Education, Lecturer on
Child Hygiene, Albany Medical College, Direc-
tor of Medico-Psychological Laboratory,
Board of Education, Albany, N. Y.

*79 Illustrations, 23 Full Page Inserts in Color and Black, and
9 Colored Text Illustrations*



PHILADELPHIA & LONDON
J. B. LIPPINCOTT COMPANY

LANE LIBRARY

COPYRIGHT, 1914
By J. B. LIPPINCOTT COMPANY

Electrotyped and Printed by J. B. Lippincott Company
The Washington Square Press, Philadelphia, U. S. A.

1914 J. B. LIPPINCOTT COMPANY

PREFACE TO PART I

THIRTY years ago even so eminent an author as Friedrich Horner considered it worth while to contribute a chapter to "Gerhardt's Manual of Diseases of Children." In that he dealt for the first time with diseases of the eye in infancy and childhood.

Ever since then this has remained a favorite and much-discussed topic in ophthalmic and pediatric investigations. In 1889, J. Michel completed the monograph of his teacher, Horner, by a systematic description of the sections that were still left untreated. In 1900, a shorter work appeared by E. Guttmann, which was followed by another contribution from A. Peters in recent times.

The extent to which the scientific discussion of these subjects has attained in the last decade will be best understood by following the annual survey of Gallus in the "Monatsschrift für Kinderheilkunde." Aside from these and numerous other sources, I have, of course, drawn upon my own practical experience of many years' standing, in dealing with the many phases this subject presents.

My desire is that the present work may not only prove useful to the general practitioner who has not made a special study of the subject treated, but also to the practical ophthalmologist as a counsellor in therapy and diagnosis.

PROF. DR. OSKAR EVERSBUCH.

MUNICH.

TRANSLATOR'S PREFACE TO PART I

THE unusual eminence which Professor Eversbusch attained among his German colleagues is sufficient reason for presenting to English readers a book written with the knowledge of an expert, and with the interest of an enthusiast.

His devotion to the subject was so great that at great personal sacrifice he became the virtual founder of what is practically an outdoor Ophthalmic Hospital for poor children at Bad Tolz, near Munich. This was done not long before the sudden and untimely end of his career. The institution, however, will remain as a monument long after this book has been outlived and forgotten.

We regret that the demands of the public upon the publishers gave us no opportunity to examine the proof sheets or make changes which are perhaps necessary.

As Prof. Eversbusch had already suggested to the older of us, that a translation of this book be made, it was therefore a pleasure to enlist the coöperation of a younger colleague, and comply with the request.

LUCIEN HOWE, M.D., Sc.D.,

Fellow of the Royal Society of Medicine.

LEE MASTEN FRANCIS, PhB., M.D., F.A.C.S.

BUFFALO, N. Y.

PREFACE TO PART II

As far back as 1750, Pelargus pointed out the impairment of the intellect occasioned by disorders of speech in childhood, without, however, drawing an erroneous parallel between development of intellect and speech. Up to 1841, the doctrine of disorders of speech occupied an important position in the realm of medical science, but since then it has received little consideration in our text-books on pediatrics. Perhaps the failure of Dieffenbach's operation for stuttering may be blamed for this. Latterly, however, medical attention has again been directed to this domain, which, though small, is important in its connection with many other sciences (pedagogy, psychology, physiology, phonetics, neurology). This reawakening is due largely to the efforts of Prof. Gutzmann, who also has assisted by word and deed in this present publication.

The following pages will attempt to derive a principal group of disorders of speech (arrests of development) from the normal development of speech, so that it is necessary to commence with a representation of the latter. A special section is devoted to those disorders of speech which children are in the habit of contracting after having acquired speech. For the sake of uniformity with the "Diseases of Children" the symptomatic disturbances in affections of the central nervous system have been treated with due regard to the contributions on this subject in Volume IV by Doctors Thiemich and Zappert.

It was necessary to go into the details of disorders of phonation, because their pathology and treatment belong to the least known subjects of pediatrics, and one must be familiar with them in order to understand the chapter on the importance and prophylaxis of disorders of speech and phonation.

As the present contribution is only part of a whole, it will be understood that frequent references to other chapters of the Handbook take the place of detailed explanations, as for instance on the causative affections, general diagnosis, treatment, etc. Other subjects, such as pneumography, should be looked up in the text-books on physiology. Having due regard to the limitations of space, it was also necessary to dispense with a reference to the complete literature and history. As far as possible, fundamental works and comprehensive explanations which embrace new or deviating view-points have been embodied.

As mentioned before, the doctrine of disorders of speech has long been neglected by official medicine. In the meantime, the treatment has fallen into the hands of charlatans, and it is time to make up our

minds to the fact that the treatment of disorders of speech belongs to the conscientious physician, who should be equipped with all the means of modern science, and that it does not belong to the domain of routine and empirical treatment, nor exclusively to that of the pedagog. Even if well-intentioned teachers honestly endeavor to exert their efforts in this direction, they should resist the temptation of encroaching upon this medical field and should not leave their special domain of pedagogy where they are in a position to render valuable assistance by coöperation within their sphere. For this and other reasons, Chapter V has been written, with special regard to the function of the school physician.

DR. MAX NADOLECZNY.

MUNICH.

TRANSLATOR'S PREFACE TO PART II

THE lack of knowledge of the more common types of speech defect on the part of general practitioners and the dearth of comprehensive works in English to which the conscientious physician might turn for help in prognosis and treatment have been evident to anyone that is familiar with the histories of any considerable number of these cases as analyzed in institutions and special schools.

The family physician too frequently dismisses the case with the assurance that the child will "outgrow the defect." As a result, in America, the "cure" of such cases has offered a rich field for the "quack."

Primary disturbances, allowed to persist, result in rapid progression of a pure defect to one that is complex in form. Valuable time is lost in some instances because of lack of knowledge of the common elocutionary methods valuable in the initial treatment of the more common defects, to say nothing of ignorance of the psychological procedures that are indicated if complete cures are to be effected.

A work so detailed from the standpoint of treatment as the one here presented should prove invaluable not only to the inquiring general practitioner but also to the specialist.

My task as English Editor of such a scholarly work has been most pleasant. It is an inspiration to be privileged to make a portion of this German lore readily available to the English reading profession.

CLINTON P. McCORD, M.D.,

Health Director, Board of Education, Lecturer on
Child Hygiene, Albany Medical College.

Director of Medico-Psychological Laboratory,
Board of Education, Albany, N. Y.

ALBANY, N. Y.

TABLE OF CONTENTS

VOLUME VII.

PART I

BY PROF. DR. OSKAR EVERSBUCH, MUNICH

DISEASES OF THE EYE IN INFANCY AND CHILDHOOD

	PAGE
I. CONGENITAL ANOMALIES AND DEFORMITIES OF THE EYE.....	1
1. Congenital Anomalies of the Lids.....	5
2. Congenital Anomalies of the Lachrymal Ducts.....	9
3. Congenital Anomalies of the Conjunctiva.....	11
4. Congenital Anomalies of the Cornea.....	14
5. Congenital Anomalies of the Uveal Tract.....	16
6. Congenital Changes of the Sclera.....	22
7. Congenital Anomalies of the Lens.....	23
8. Persisting Remains of the Tunica Vasculosa Lentis.....	27
9. Congenital Anomalies of the Vitreous.....	31
10. Congenital and Infantile Glaucoma (Hydrophthalmos Congenitus).....	33
11. Congenital Anomalies and Deformities of the Optic Nerve and Retina...	35
12. Congenital Anomalies of the Orbit.....	38
13. Congenital Anomalies of Pigmentation.....	40
14. Congenital Disorders of the Ocular Nerves and Muscles.....	41
15. Ocular Anomalies or Affections Due to Ancestry, Heredity, or Consanguinity.....	44
II. DEVELOPMENT OF THE INFANTILE EYE.....	49
III. EXAMINATION OF CHILDREN WITH OCULAR AFFECTIONS.....	63
IV. AFFECTIONS OF THE EYELIDS.....	67
1. Spontaneous (Non-traumatic) Hemorrhage and Circulatory Disease of the Lids.....	69
2. Variola of the Lids.....	71
3. Vaccine Infection of the Lids.....	74
4. Erysipelas of the Lids.....	76
5. Anthrax Infection of the Lids.....	77
6. Gangrene of the Lids.....	78
7. Phlegmonous Inflammations and Abscesses of the Lids.....	79
8. Herpes of the Lids.....	80
9. Tuberculosis of the Lids.....	81
10. Syphilitic Affections.....	82
11. Soft Chancre.....	86
12. Glanders.....	87
13. Actinomycosis.....	87
14. Leprosy.....	88
15. Eczema of the Lids.....	89
16. Phthiriasis Palpebralis.....	93
17. Hordeolum.....	94
18. Chalazion.....	95
19. Faulty Position of the Cilia (Trichiasis and Distichiasis).....	97
20. Blepharospasm.....	99
21. Lagophthalmos.....	103
22. Blepharophimosis.....	105
23. Entropion.....	105

24. Ectropion.....	106
25. Affections of the Levator Palpebræ.....	107
26. Tumors.....	109
V. AFFECTIONS OF THE LACHRYMAL ORGANS.....	110
1. Inflammation of the Lachrymal Glands.....	111
2. Partial or Total Arrest of Lachrymation.....	113
3. Epiphora (Pathological Lachrymation).....	113
4. Blennorrhœa of the Lachrymal Canals.....	118
5. Acute (Phlegmonous) Dacryocystitis and Blennorrhœa of the Lachrymal Sac.....	118
VI. AFFECTIONS OF THE CONJUNCTIVA.....	125
1. Hyperæmia and Catarrh of the Conjunctiva.....	127
2. Eczematous Conjunctivitis and Keratitis.....	136
3. Variolar Pustules of the Conjunctiva.....	145
4. Vaccinola and Varicellæ.....	146
5. Croup and Diphtheria of the Conjunctiva.....	146
6. Pemphigus.....	152
7. Gonorrhœal Ophthalmia.....	153
8. Trachoma.....	169
9. Conjunctival Folliculosis; Follicular Catarrh.....	178
10. Syphilitic Affections of the Conjunctiva.....	179
11. Tuberculosis of the Conjunctiva.....	182
12. Leprous Affections of the Conjunctiva.....	184
13. Spring Catarrh.....	184
14. Lymphoma of the Conjunctiva.....	186
15. Epithelial Xerosis of the Conjunctiva.....	186
16. Pseudopterygium.....	187
17. Circulatory Disturbances and Hemorrhages.....	187
18. New Growths of the Conjunctiva.....	189
VII. AFFECTIONS OF THE CORNEA.....	191
1. Herpes of the Cornea.....	194
2. Keratitis Disciformis.....	198
3. Eczematous Ulcers of the Cornea.....	198
4. Pannus of the Cornea.....	202
5. Marantic Necrosis of the Cornea.....	203
6. Keratitis Following Lagophthalmos.....	205
7. Keratitis Neuroparalytica.....	206
8. Septic Suppurative Keratitis.....	207
9. Parenchymatous Keratitis.....	211
10. Corneal Changes in Leprosy.....	223
11. Keratoconus.....	224
VIII. AFFECTIONS OF THE SCLERA.....	225
IX. DISEASES OF THE UVEAL TRACT.....	230
1. Syphilitic Affections.....	236
2. Uveal Tuberculosis.....	244
3. Hemeralopia.....	248
4. Uveal Affections in Leprosy.....	250
5. Recurring Vitreous Hemorrhage.....	251
6. Acute Metastatic Suppurative Iridochorioiditis.....	252
7. Uveal Tumors.....	256
X. AFFECTIONS OF THE LENS.....	257
XI. GLAUCOMA.....	261
XII. AFFECTIONS OF THE OPTIC NERVE AND RETINA.....	263
1. Hereditary Retrobulbar Optic Neuritis.....	265
2. Pigment Degeneration of the Retina.....	265
3. Simple Atrophy of the Optic Nerve.....	266
4. Visual Disturbances Due to Loss of Blood.....	268
5. Peripheral Neuritis of the Optic Nerve.....	269
6. Toxic Amblyopia.....	274
7. Syphilis.....	274

TABLE OF CONTENTS

xi

8. Albuminuric Retinitis.....	275
9. Choked Disc.....	279
10. Changes of the Optic Nerve in Oxycephalia.....	280
11. Glioma of the Retina.....	281
12. Other Tumors of the Optic Nerve.....	283
XIII. ANOMALIES OF REFRACTION AND ACCOMMODATION.....	285
1. Hypermetropia.....	289
2. Myopia.....	290
3. Astigmatism.....	298
4. Disturbances of Accommodation.....	300
5. Paralysis of Accommodation.....	301
XIV. DISTURBANCES OF PUPILLARY MOVEMENTS.....	305
XV. AFFECTIONS OF THE EXTERNAL MUSCLES OF THE EYE.....	310
1. Asthenopia.....	310
2. Concomitant Squint.....	311
3. Irritation and Spasm of the External Ocular Muscles.....	316
4. Paralysis of the Ocular Muscles.....	317
XVI. DISEASES OF THE ORBIT.....	321
1. Inflammations of the Orbit.....	321
2. Serous Infiltration and Phlegmon of the Orbit.....	325
3. Orbital Periostitis.....	326
4. Tuberculosis of the Orbit.....	327
5. Orbital Hemorrhages.....	329
6. Neoplasms of the Orbit.....	329
XVII. INJURIES OF THE EYE.....	331
1. Foreign Bodies in the Conjunctiva, Lachrymal Passages, and Cornea....	331
2. Ophthalmia Due to Caterpillar Hairs.....	336
3. Foreign Bodies in the Interior of the Eye.....	339
4. External Injuries of the Eye.....	343
5. Burns and Corrosions of the Conjunctiva and Cornea.....	345
6. Cuts, Stabs, and Tears of the Eyeball.....	347
7. Contusions of the Eye.....	349
8. Injuries of the Orbit.....	353
9. Sympathetic Ophthalmia.....	356

PART II

BY DR. MAX NADOLECZNY, MUNICH

DISORDERS OF SPEECH AND PHONATION IN CHILDHOOD

I. PSYCHOLOGY AND PHYSIOLOGY OF THE DEVELOPMENT OF SPEECH.....	359
1. Preliminary Conditions for the Development of Speech.....	359
1. The Acustico-optical Development.....	359
2. The Motor-kinæsthetic Development.....	360
3. The Idiomatic Development.....	361
4. The General Physico-psychic Development.....	361
2. Preliminary Stages of Lingual Development.....	361
3. The First Independent Talking.....	366
4. Formation of Sentences.....	373
5. Differences in Development.....	375
6. Physiology of the Complete Language.....	376
II. LINGUAL ARRESTS OF DEVELOPMENT.....	378
1. Peripherally Impressive Arrests.....	378
2. Central Arrest of Lingual Development.....	387
Lingual Deafness (Sensory Aphasia).....	387
Auditory Dumbness (Audimutitas).....	388
Voluntary Whispering.....	391
Spluttering (Paraphrasia Præceps).....	392
Akataphasia and Agrammatism.....	394
Functional Stammering (Dyslalia).....	396

TABLE OF CONTENTS

Forms of Functional Stammering.....	397
1. Vowel-stammering	397
2. Stammering in Labial Sounds.....	397
3. Stammering in Dental Sounds.....	397
4. Stammering in Palatal Sounds (Gammacism).....	397
5. L-stammering.....	398
6. R-stammering.....	398
7. Syllable- and Word-stammering.....	398
3. Peripheral Expressive Arrests of Lingual Development.....	399
Lisping.....	400
1. Strident Sigmatism.....	401
2. Addental Sigmatism.....	401
3. Interdental Sigmatism.....	401
4. Labio-dental Sigmatism.....	401
5. Lateral Sigmatism.....	402
6. Nasal Sigmatism.....	402
7. Parasigmatism.....	402
Snuffling (Rhinolalia).....	405
1. Rhinolalia Clausa.....	406
2. Rhinolalia Aperta.....	406
3. Rhinolalia Mixta.....	408
III. DISORDERS OF SPEECH.....	412
1. Stuttering (Balbuties).....	412
2. Disorders of Speech at Puberty.....	434
3. Disorders of Speech in Functional Diseases of the Nervous System.....	436
4. Disorders of Speech in Chorea Minor.....	436
5. Disorders of Speech in Epilepsy.....	436
6. Disorders of Speech in Maladie des Tics (Facial Spasms).....	437
7. Disorders of Speech in Hysteria.....	438
8. Congenital and Early Acquired Disorders of Speech.....	440
9. Mutism of Imbeciles.....	441
10. Disorders of Speech in Partially Deaf Imbeciles.....	446
11. Echolalia.....	447
12. Stammering of Imbeciles.....	448
13. Akataphasia of the Imbecile.....	449
14. Stuttering of the Imbecile.....	449
15. Disturbance of Lingual Accents in the Imbecile.....	450
16. Disorders of Speech Owing to Organic Affections of the Nervous System.....	451
17. Disorders of Speech Owing to Hereditary Syphilis of the Central Nervous System.....	453
18. Disorders of Speech Due to Encephalitis.....	455
19. Disorders of Speech Due to Sclerosis of the Central Nervous System.....	455
20. Disorders of Speech Due to Infantile Cerebral Paralysis.....	456
21. Disorders of Speech Due to Affections of the Peripheral Nervous System and the Muscular Apparatus.....	459
Disorders of Speech Due to Paralysis of the Facial.....	459
Mutism and Aphasia in Childhood.....	459
1. Mutitas Physiologica.....	459
2. Mutitas Prolongata and Audimutitas.....	460
3. Mutitas Prolongata.....	460
4. Mutitas Idiotica.....	460
5. Mutismus Hystericus.....	460
6. Aphasia.....	460
7. Aphasia Voluntaria.....	462
IV. DISORDERS OF THE VOICE.....	463
1. Physiology of the Infantile Voice.....	463
2. Disturbances of the Voice.....	466
V. IMPORTANCE AND PROPHYLAXIS OF DISORDERS OF SPEECH AND VOICE IN SCHOOL.....	469
VI. THE INABILITY TO LEARN TO READ. SO-CALLED CONGENITAL WORD-BLINDNESS.....	476
LITERATURE.....	479

LIST OF ILLUSTRATIONS

VOLUME VII

	PAGE
1. CONGENITAL PTOSIS AND EPICANTHUS.....	5
2. CRYPTOPHTHALMOS.....	7
3. EWETZKY'S FOLD.....	13
4. COLOBOMA OF THE CHOROID. (AFTER ED. V. JAEGER).....	17
5. COLOBOMA OF THE OPTIC DISC.....	17
6. PERSISTING PUPILLARY MEMBRANE. (AFTER E. v. HIPPEL).....	28
7. HYPERPLASIA OF THE INFERO-INTERIOR SECTOR OF THE IRIS.....	30
8. PERSISTING HYALOID ARTERY WITH CONDENSED GLIAL COAT.....	32
9. CONGENITAL HYDROPHTHALMOS OF THE LEFT EYE.....	34
10. PREPAPILLARY CONNECTIVE TISSUE.....	37
11. DERMOID OF THE ORBIT WITH EXOPHTHALMOS AND DISPLACEMENT OF THE BULB DOWNWARD.....	39
12. INSTILLATION.....	65
13. PTOSIS IN TRACHOMA.....	68
14. DILATED LID FISSURE IN EXOPHTHALMOS.....	68
15. BLEPHAROSPASM IN SCROFULOUS OPHTHALMIA.....	68
16. THERMOPHORE.....	73
17. JAPANESE WARMING BOX.....	73
18. GANGRENE OF THE LID IN VACCINE OPHTHALMIA.....	79
19. BLEPHAROSPASM.....	100
20. INCISION OF THE OUTER COMMISSURE OF THE LID.....	101
21. POSITION OF THE SUTURES IN CANTHOPLASTY.....	101
22. NORMAL PALPEBRAL FISSURE.....	102
23. LID FISSURE AFTER CANTHOPLASTY.....	103
24. THE INSTILLATION OF DROPS.....	133
25. HABITUS SCROFULOSUS.....	137
26. THE INSUFFLATION OF POWDER IN THE EYE.....	144
27. WATCH-GLASS BANDAGE.....	168
28. VARIOUS TYPES OF HERPES CORNEÆ.....	194
29. ILLUMINATING LENS.....	195
30. LINEAL CAUTERIZATION OF THE LOWER FORNIX.....	205
31. BINOCULAR BANDAGE.....	209
32. HUTCHINSON'S TEETH.....	216
33. INSERTION OF AN ARTIFICIAL EYE.....	254
34. REMOVAL OF AN ARTIFICIAL EYE.....	255
35. IMPROVED ("REFORM") EYE.....	255
36. EYE SCREEN.....	260
37. SUCTION APPARATUS IN OPERATION.....	273
38. CONVERGENT STRABISMUS.....	313
39. DIVERGENT STRABISMUS.....	313
40. PARESIS OF THE RIGHT OCULOMOTORIUS NERVE SINCE HER THIRD MONTH, IN 18-MONTHS-OLD CHILD. NO LUES.....	318

41. LEUKÆMIC TUMORS OF THE ORBIT.....	328
42. EYE PAD.....	333
43. REMOVAL OF A FOREIGN BODY FROM THE CORNEA.....	335
44. MONOCULAR BANDAGE.....	335
45. ECTROPION OF THE RIGHT UPPER LID OF THE EYE AFTER A BURN.....	345
46. SYMBLEPHARON AFTER LIME BURN.....	346
47. INFANTILE CRY-CURVE.....	360
48. DISTRIBUTION OF WORD CATEGORIES DURING THE DEVELOPMENT OF SPEECH OF DEVILLE'S DAUGHTER IN PER CENT.....	371
49. QUANTITATIVE INCREASE OF VOCABULARY OF DEVILLE'S DAUGHTER FROM ONE YEAR SEVEN MONTHS TO THE END OF THE SECOND YEAR.....	371
50. READING AND BREATHING CURVE OF A THIRTEEN-YEAR-OLD HEALTHY GIRL.....	376
51. DIAGRAM OF A GLOTTIS AT THE ONSET OF A SOFT SOUND, THE INFANTILE FORM BEING CONSIDERED.....	377
52. DIAGRAM OF A GLOTTIS AT THE ONSET OF A HARD SOUND, BEFORE PROTRUSION OF THE VOCAL CORDS.....	377
53. DIAGRAM OF A GLOTTIS, WHILE WHISPERING.....	377
54. WELL-DEVELOPED, NORMAL BOY OF FIFTEEN.....	380
55. BOY OF THIRTEEN, DEAF SINCE 1901 AFTER SCARLET FEVER.....	381
56. NUMBER OF WORDS (FINGER ALPHABET).....	385
57. BOY WITH AUDITORY DUMBNESS.....	390
58. FORMATION OF THE K-SOUND.....	398
59. POSITION OF THE TIP OF THE TONGUE FOR THE CORRECT PRONUNCIATION OF S, WITH THE AID OF GUTZMANN'S SOUND.....	404
60. PUSHING THE TONGUE BACKWARD WITH THE RING-SOUND TO FORM THE SH-SOUND.....	404
61. THE VELUM PALATI IN THE POSITION OF RESPIRATION.....	405
62. SPEAKING AND RESPIRATION CURVE IN RHINOLALIA APERTA.....	407
63. NASAL CURVE IN RHINOLALIA APERTA.....	409
64. POSITION OF GUTZMANN'S HAND OBTURATOR AGAINST THE SOFT PALATE.....	411
65. HEREDITARY TAINT.....	414
66. APPARENT (HATCHED) AND GENUINE (BLACK) TRANSMISSION OF STUTTERING....	414
67. CURVE OF AIR GUSTS, ESCAPING THROUGH THE NOSE IN CLONIC SPASMS OF THE VELUM PALATI.....	419
68. SPASM ON PRONOUNCING B.....	422
69. SPASM ON PRONOUNCING D.....	422
70. HEMIHYPOLASIA OF THE FACE.....	424
71. CORRECT BREATHING EXERCISE.....	429
72. WRONG BREATHING EXERCISE.....	429
73. WRONG RESPIRATORY VOCAL EXERCISE.....	430
74. AVERAGE RANGE OF BOYS' VOICES AND GIRLS' VOICES FROM 0 TO 15 YEARS.....	464
75. DIAGRAM OF THE GLOTTIS FISSURE ON SOUNDING THE WHISTLING REGISTER....	465
76. DIAGRAM OF THE GLOTTIS FISSURE IN MUTATION.....	465
77. FIGURE SHOWING THE PERCENTAGE OF STAMMERERS OF A CERTAIN AGE, THE DECREASE OF STAMMERING DURING SCHOOL AGE BEING VERY RAPID.....	469
78. FIGURE SHOWING HOW MANY OF THE 100 STUTTERERS OF THE OLDER GRADE HAVE DROPPED OUT.....	470
79. CHRONIC HOARSENESS IN 575 SCHOOL CHILDREN OF BOTH SEXES, COMPILED ACCORDING TO PERCENTAGE AND AGE.....	473

LIST OF PLATES

PLATE I.....	4
Fig. 1. Coloboma of the Lid.	
Fig. 2. Dermoid with Embryontoxon.	
Fig. 3. Congenital Coloboma of the Iris.	
Fig. 4. Ulcerative Blepharitis. Partial Madarosis.	
Fig. 5. Squamous Blepharitis.	
Fig. 6. Hordeolum.	
PLATE II.....	70
Fig. 1. Abscess on Upper Lid.	
Fig. 2. Chalazion.	
Fig. 3. Vaccine Pustule on Lower Lid.	
Fig. 4. Herpes Zoster Ophthalmicus.	
Fig. 5. Dacrocystitis Phlegmonosa.	
PLATE III.....	126
Fig. 1. Conjunctival Injection.	
Fig. 2. Slough after Lineal Cauterization of the Transitional Fold.	
Fig. 3. Effect of a 5 per cent. Dionin Instillation.	
Fig. 4. Ecchymosis of the Conjunctiva in Pertussis.	
PLATE IV.....	128
Fig. 1. Diplobacilli (Morax-Axenfeld).	
Fig. 2. Pneumococci.	
Fig. 3. Xerosis Bacilli.	
Fig. 4. Gonococci.	
PLATE V.....	136
Fig. 1. Blepharoconjunctivitis (Morax-Axenfeld).	
Fig. 2. Phlyctenular Conjunctivitis.	
Fig. 3. Croupous Conjunctivitis.	
Fig. 4. Blepharitis Neonatorum.	
Fig. 5. Folliculosis.	
Fig. 6. Fresh Trachoma.	
PLATE VI.....	138
Fig. 1. Cicatricial Trachoma.	
Fig. 2. Spring Catarrh.	
Fig. 3. Leucoma Corneæ Adhærens.	
Fig. 4. Corneal Ulcer. Corneal Macula.	
Fig. 5. Total Staphyloma.	
Fig. 6. Keratitis Fascicularis.	
PLATE VII.....	140
Fig. 1. Prolapse of Iris after Perforation of a Phlyctenular Marginal Ulcer.	
Fig. 2. Pannus Keratitis.	
Fig. 3. Serpiginous Ulcer of the Cornea with Hypopyon.	
Fig. 4. Keratitis Disciformis (Post-vaccinosa).	
Fig. 5. Parenchymatous Keratitis.	
Fig. 6. Avascular Parenchymatous Keratitis.	
Fig. 7. Deep Vessels in Parenchymatous Keratitis.	
PLATE VIII.....	226
Fig. 1. Scleral Staphyloma in Intraocular Tumor.	
Fig. 2. Hamburger's Tentative Treatment of Iritis.	
Fig. 3. Pericorneal Conjunctival and Subconjunctival Injection. Iridodialysis.	
Fig. 4. Tubercle in the Angle of the Anterior Chamber.	
Fig. 5. Pseudoglioma.	
PLATE IX.....	258
Fig. 1. Total Congenital Cataract.	
Fig. 2. Persistent Hyaloid Artery Adherent to the Posterior Capsule of the Lens.	
Fig. 3. Laminated Cataract.	

Fig. 4.	Laminated Cataract.	
Fig. 5.	Congenital Dislocation of the Lens (Focal Illumination).	
Fig. 6.	Congenital Dislocation of the Lens (Translumination).	
PLATE X.....		260
Fig. 1.	Exophthalmos Due to Retrobulbar Effusion Owing to Contusion.	
Fig. 2.	Foreign Body on the Conjunctiva of the Upper Lid.	
Fig. 3.	Lime Corrosion of the Conjunctiva and Cornea.	
Fig. 4.	Pericorneal Ciliary Injection. Corneal Scar with Adhesion to the Iris.	
Fig. 5.	Blood Effusion into the Anterior Chamber in Contusion of the Bulb.	
PLATE XI.....		264 ✓
	Normal Ocular Fundus.	
PLATE XII.....		266
	Physiological Excavation.	
	Scleral Ring with Pigment Ring.	
PLATE XIII.....		268
	Conus Inferior.	
	Choked Disk.	
PLATE XIV.....		270
	Genuine Atrophy of the Optic Nerve.	
	Neuritic Atrophy of the Optic Nerve.	
PLATE XV.....		272
	Glaucomatous Excavation.	
	Albuminuric Neuroretinitis.	
PLATE XVI.....		278
	Retinitis Pigmentosa.	
	Disseminated Choroiditis.	
PLATE XVII.....		282
	Congenital Syphilitic Chorioretinitis.	
	Circumpapillary Atrophy of the Choroid (Myopia).	
PLATE XVIII.....		290
	Central Chorioretinitis.	
	Oblique Position of the Opticus, Temporal Conus (Myopia).	
PLATE XIX.....		294
	So-called "Coloboma" of the Macula Lutea.	
PLATE XX.....		318
	On the Right, the Nerves; on the Left, the Vessels of the Orbits.	
PLATE XXI.....		334
Fig. 1.	Strabismus Hook.	
Fig. 2.	Discission Needle.	
Fig. 3.	Græfe's Cataract Knife.	
Fig. 4.	Weber's Lachrymal Knife.	
Fig. 5.	Conical Sound.	
Fig. 6.	Bowman's Sound.	
Fig. 7.	Lachrymal Sac Syringe. (After Anel.)	
Fig. 8.	Ciliary Forceps.	
Fig. 9.	Iris Forceps.	
Fig. 10.	Galvanocautery Syringe.	
PLATE XXII.....		400
Fig. 1.	High Degree of Open Dentition. Interdental Sigmatism.	
Fig. 2.	Addental Sigmatism.	
Fig. 3.	Cross-dentition. Lateral Sigmatism.	
Fig. 4.	Lateral Gaps in Abnormal Dental Arches. Lateral Sigmatism.	
PLATE XXIII.....		418
	Figs. 1 to 6. Speaking Curves of Stutterers.	

THE DISEASES OF CHILDREN

DISEASES OF THE EYE IN INFANCY AND CHILDHOOD

I. CONGENITAL ANOMALIES AND DEFORMITIES OF THE EYE

IN these pages congenital defects of the eye are discussed only in so far as they are of clinical importance to ophthalmologists from the stand-point of differential diagnosis.

The development of the eye, in which the optic vesicle, the germinal vesicle of the lens, the meso- and ectoderm tissues participate, takes the following course: The first visible rudiment consists of the two optic grooves, which are situated in the wall of the primitive cerebrum, on either side of the median line, and tend to turn downward as the central medulla is closed.

The optic vesicles, which were formerly termed "primary optic vesicles," are formed by a protrusion of the cerebrum—this part being united to the ventricle by a short pedicle. This becomes the foundation of the retina and the pigment epithelium, while the optic nerves are formed out of the pedicle. The embryonic eye, or optic cup ("secondary optic vesicle"), develops from the extending margin, which gradually encloses the original base of the optic vesicle. The fundus is now provided with two walls: the inner layer or retina, and the outer layer or pigment epithelium. On its ventral side it contains the fetal optic fissure, which is continued to the pedicle. The margins of the latter approach each other, until, at the end of the first month, they fuse.

The optic cup is scoop-shaped, the dorsal retroverted margin of its two layers being much more distally situated than the ventral one.

The transformation of the optic vessel is independent of the development of the germinal vesicle of the lens, which occupies the largest portion of the distal opening of the optic cup. At first it is merely a hollow indentation of the ectoderm which is thickened at a circumscribed place, the latter advancing toward the fossicular indentation formed by the cup.

The germinal vesicle of the lens, which is connected at first with the surface by a pedicle, is later separated from it and then the ectoderm grows over it. The mesoderm, from which there are developed the fibrillar stroma of the vitreous, the chorioid, sclera, the connective tissue, part of the corpus ciliare, iris, ligamentum pectinatum, substantia pro-

pria of the cornea, and the optic vessels, grows across the optic cup in such a way that its "intra-ocular" part, or the so-called tunica vasculosa lentis, remains in connection with the part surrounding the optic cup through the fetal optic fissure until the time of its closure, and also through the place anterior to the retroverted margin of the optic cup.

As for further details, these take place about as follows: The separation of the retina into its future layers begins in the region of the future fovea. The delicate nerve-fibres, which are formed early, penetrate centripetally from the retina into the pedicle of the optic vesicle. The rods, cones, and fovea centralis are formed last. In the new-born the cones of the fovea centralis are still incomplete.

The fibres of the zonula are formed from the cells of the ciliary part of the retina, the double epithelial layer of the ciliary body and iris from the retroverted margin of the optic cup, and the sphincter iridis (at the end of four months) from the plica iridica retinæ. The mesodermal part of the iris is a continuation of the chorioid. The pupillary margin, which, at the end of six months, lies but slightly inside of the lenticular equator, has arrived at its definite place in the seventh month.

The development of the retinal vessels from the arteries of the optic and retinal nerves takes place between the third and eighth months. The arteria centralis, which is already enclosed in the optic nerve at the close of the optic fissure, advances later from the periphery to the axis of the nerve.

The vitreous body is composed chiefly of fibrillæ, which are attached to the inner surface of the retina, form numerous transverse anastomoses and radiate toward the inner surface of the lens. These fibres, which are supported by a stronger fibrous tract from the margin of the optic vessel, connect with the walls of the vessels permeating the embryonal vitreous.

The cells of the proximal wall of the lenticular germinal vesicle develop into lenticular fibres, those of the distal wall into capsule epithelium. Pigmentation of the chorioid, which commences very early with the inner layers of the sclera in a rudimentary way, does not occur until after the fifth month. It is still very slight at birth, if present at all. The ciliary processes develop from radial foldings of the distal part of the wall of the optic cup from the tenth to the twelfth week, the deeper parts of the folds being filled up by the surrounding mesoderm.

About the eighth or ninth week there is a separation of the rudimentary mesoderm, which has penetrated between the ectoderm (the future corneal epithelium) and the lens, into the substance proper of the cornea, the corneal endothelium, ligamentum pectinatum, and pupillary membrane. The development of the anterior chamber, which is at first visible as an annular fissure in the peripheral parts, becomes well

defined in the fifth or sixth month. Its full development is not reached until maturity is attained.

The tunica vasculosa is not only important for the structure of the eye and the nutrition of the growing lens, but also for the formation of the vitreous. While the anterior portion, the membrana pupillaris, is connected with the mesoderm layer which envelops the entire eye, the posterior portion, which springs from the central artery of the primordial retina, is continued into the posterior part of the vascular lenticular surface. This area contains the central artery of the vitreous or hyaloid body with its branches at the posterior lenticular surface, and the vasa hyaloidea propria, which runs through the peripheral parts of the vitreous. Some of its branches also run to the pupillary membrane, while others, turning sharply around the margin of the optic cup, fuse directly with the veins of the iris. Besides, the anterior part of the tunica vasculosa lentis is supplied with blood from the arteries emanating from the long ciliary arteries, and passing from the iris into the pupillary membrane. The blood from the latter and the iris is then supplied to the venæ vorticosæ.

The vasa hyaloidea propria undergo involution between the third and sixth months, while a remnant of the hyaloid artery, measuring 1 to $1\frac{1}{2}$ mm., is still present in the new-born.

The branches of the hyaloid artery and the posterior parts of the tunica vasculosa of the lens disappear sooner than the pupillary membrane. The involution of the latter commences in the eighth month, terminating with the obliteration of the peripheral parts, which extend to the region of the small circle of the iris.

The eyelids are formed from skin folds, the inner surface of which will later form the conjunctiva. The growing folds approach each other above and below the cornea, their free margins fusing in the third month by means of an epithelial layer which disappears before birth.

The lachrymal gland is formed by a proliferation of the conjunctival epithelium. The lachrymal duct, by an epithelial germination which, separating itself from the cuticle surface, becomes hollow as growth proceeds, connects upward with the conjunctival sac through the lachrymal tubules (which also originated from this epithelial cord) and downward with the nasal cavity.

The lower end of the naso-lachrymal duct, however, does not become permeable until a relatively later period, and may still be occluded by a fine membrane at the time of birth.

The somewhat complicated development of the orbit, the optic muscles and nerves, the lids, conjunctival sac, the glandular apparatus of the conjunctiva, etc., is more or less independent of that of the bulb, so that the former may be fully developed, when the bulb is still absent.

The immediate cause of congenital defects of the globe and of its accessory parts is not yet entirely cleared up, but it is known from a study of the history of development and anatomy of abnormal processes that, for instance, abnormal development of the mesodermal tissue may not only prevent the closure of the fissure of the optic cup but also cause typical anomalies in the internal eye. Still more important is v. Szily's investigation, according to which a germinal variation of the ectodermal rudiments may be responsible for the occurrence of coloboma in the fundus. Another point is that the latest possible time for the production of many malformations has been definitely determined. This has been called the teratogenetic period of germination.

The remote cause is less clear; but it is very probable that a variation of germ plasm or a faulty condition of the germ (egg or spermatozoa) is an "internal" cause in anomalies of development and affections of the eye which have been inherited in the course of several generations. This refers to laminated or central cataract, incomplete closure of the optic vessel, albinism, buphthalmos, congenital opacity of the cornea, anisometropia, high degrees of congenital myopia, unilateral amblyopia, corneal astigmatism with pathological defects of other parts of the eye, nystagmus, unilateral and high degrees of hypermetropia, retinitis pigmentosa, congenital stationary night-blindness, congenital paralytical strabismus, concomitant strabismus due to congenitally defective fusion, strabismus either congenital or due to congenital insufficiency of the macular region, chorioretinitis, etc.

The ocular defects may vary in different generations, as for instance coloboma in the first generation, iridderemia and other defects in the next. Similarly, the form of congenital cataract may vary in the same family, appearing either as typical laminar cataract, or as fusiform, polar or central cataract, and, besides, may occur at earlier periods in each following generation.

By poisoning pregnant rabbits with naphthalin, Pagenstecher produced congenital cataract and ocular malformations.

Anomalies caused by arrest of lid formation or destruction of the formed lid, palpebral coloboma, epi- and circumbulbar dermoids of the bulb, and cryptophthalmos, have been traced to "external" causes, such as extreme narrowness of the amniotic sac or mechanical effects of the amniotic cord, the variable force of the latter explaining the variability of the deformities. Other amniotic anomalies, such as syndactylism of the hands or feet, deformation of the genitals, etc., often complicate the manifestations.

True deformities of the eye are usually pure arrests of development, and occur in the earliest period of extra-uterine life, as, for instance, at the end of the first month, that being the time when the optic fissure

PLATE I.



FIG. 1. Carbuncle of the lid.



FIG. 2. Dermoid with embryonic tooth.



FIG. 3. Congenital coloboma of the iris.



FIG. 4. Ulcerative Ophthalmia. Partial mastareosis.



FIG. 5. Squamous ophthalmia.



FIG. 6. Hordeolum.

closes. Fetal defects of the eye, however, which resemble extra-uterine inflammation of the eye in many respects, usually develop toward the end of embryonal life. At the same time, it cannot always be definitely stated whether an existing condition is essentially a malformation or the result of a fetal affection, or a combination of both.

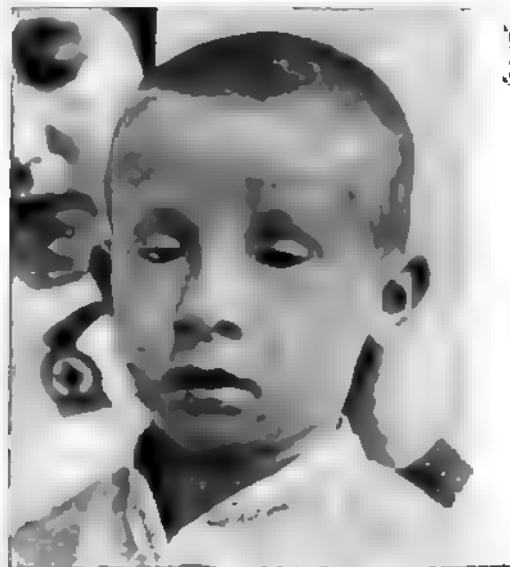
The anomalies caused by fetal inflammation are often clinically complicated cases. Zimmermann, for instance, found in an atypical coloboma of the iris signs of perforation of the limbus and irregular demarcation of the cornea.

So-called signs of degeneration have been found in imbecile and psychopathically affected infants, such as coloboma of the lids, uvea, conus inferior of the optic disc, heterochromia iridis, pigmental spots in the iris, excentric or oval pupils, albinism, medullated nerve-fibres, epicanthus, and persistent hyaloid artery.

1. CONGENITAL ANOMALIES OF THE LIDS

Shortening of the lid fissure ordinarily occurs with anomalies of development, such as bilateral ptosis (Fig. 1), flat nasal bridge, flat facial contour, and mental inferiority (Mongolism). Von Michel also

Fig. 1.



Congenital ptosis and epicanthus.

observed mixed astigmatism, punctiform opacities of the lens, and hereditary syphilitic changes of the peripheral chorioidal vessels. Abnormally short upper lids are sometimes found in congenital ptosis.

Congenital coloboma of the lid (Plate I, Fig. 1) appears as a V-shaped or rounded notch, with its base toward the margin of the lid,

the upper lid being more frequently affected than the lower one. It varies in size, and may occur simultaneously in one or more lids and, similarly, several defects may be present in any one lid. The central portion of the lid is most frequently involved. There are often accompanying changes of the surrounding parts, such as cicatricial furrows running to the region of the eyebrows. The latter may be partly absent, or the hair line may be lost in the frontal region. Further defects found are conjunctival folds, pterygium-like formations, subconjunctival and epibulbar dermoids and lipodermoids; folds of the skin, which arising from the notch extend to the globe, thus connecting the coloboma with the cornea and sclera, and thereby causing disturbances of motility and vision. Such, for example, are opacities of the cornea, symblepharon, absence of caruncles or of a complete external canthus, supernumerary puncta lacrimalia, or their absence, or open lachrymal sac and duct. Of less frequent occurrence are anomalies of bulbar formation, such as microphthalmos, corectopia, uveal coloboma, persistent pupillary membrane, etc. Malformations of the face and buccal cavity are of greater frequency, such as harelip and cleft palate; cerebral anomalies like exencephaly, hemiccephaly, anencephaly; or they may occur in other parts of the body, such as abdominal rupture, syndactylism, or spontaneous amputation of parts of the extremities. In some cases the lid coloboma appears as a prolongation of the so-called oblique facial fissure, which is due to defective union of the maxillary processes and the frontal bone. Atypical folds of the superior maxilla may be prolonged into the lid.

Treatment.—The coloboma may be removed by blepharoplastic surgery. Otherwise the eye should be protected by special spectacles.

Dermoids or dermoid cysts occurring at the junction of the superior maxilla and the frontal process (that is, at the inner canthus or middle half of the upper lid), or occurring at a point corresponding to the suture zygomatico frontalis (that is, at the external canal or lateral superior orbital margin), are usually connected at their bases with the periosteum by means of a pedicle. This last may reach far into the orbit, having some former relation to the deep sutures. Since they continue to extend and sometimes suppurate, it is necessary to resort to surgical removal.

The same applies to verrucose hair marks (*nævi pilosi*) and pigmented *nævi* which often develop later into melanosarcoma; also to lipoma, angio-, fibro-, or neurolipoma, to encysted lymphangioma, and to the various combination forms of neurofibroma of the lids (plexiform neurofibroma, fibroma molluscum, unilateral facial hypertrophy). On the other hand, in angiomas which develop superficially or subcutaneously (*hæmangioma simplex*, *plexiforme*, and *cavernosum*), diffuse lymphangioma simplex and cavernosum, provided they are not too extensive, electrolysis often causes a fibromatous degeneration and gradual

reduction of the growth in such a satisfactory manner that purely surgical treatment may be discarded.

Treatment.—The positive pole, consisting of a moist pad, is applied to the nape of the neck, and a needle attached to the negative pole of a direct current of 2 milliampères is inserted flat into the growth for one minute. This treatment is repeated two or three times, the needle being inserted in a different part on each occasion. Several treatments are usually required. Local anæsthesia, if necessary, is best effected with eusemin (75 per cent. cocaine and 0.05 per cent. adrenalin).

In *cryptophthalmia*, either congenital (in a single individual) or as a family affection, one lid fissure is usually completely missing. Cases with small remnants of the conjunctival sac or a considerable part of it are of rare occurrence (Fig. 2). The skin, extending uninterruptedly from the forehead to the cheek, completely covers the globes, which, however, can be felt and are (usually) apparently movable. This deformity is caused by the fact that the mesoderm and ectoderm, growing over the optic vessel, formed skin instead of the cornea. This accounts for the arrested development of lids and conjunctival sac (E. v. Hippel). Experience has shown that operative procedure is quite useless, and, besides, anatomical examinations have established the fact that the development of the cornea has been arrested, or that there is absence or rudimentary development of the lens and signs of chronic inflammation of the globe, especially in its deeper parts.

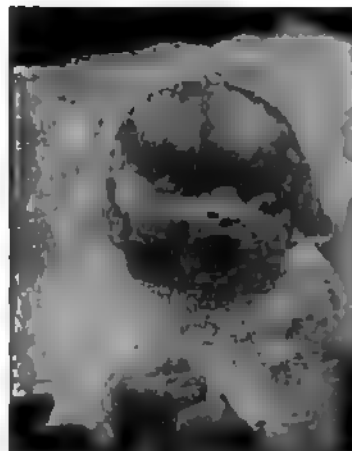


FIG. 2

Cryptophthalmos.

The so-called *Mongolian fold* is a crescent-shaped fold of skin bent outward, which extends toward the median line from the inner canthus to the root of the nose. There it fuses with a transverse fold of the upper lid (Kollmann's *plica marginalis*). This is a mild degree of what v. Ammon named *epicanthus* (Fig. 1) and is quite common in children of certain localities.

This anomaly of development usually disappears as the growth of the bridge of the nose progresses.

This fold is more strongly developed in the Mongolian race, and causes the peculiar obliquity of the lid. It may be made to disappear by pinching up the skin at the root of the nose into a longitudinal fold. The *lacus lacrimalis* is usually completely covered by this fold.

The fully developed congenital "*epicanthus*" (*supraciliaris*, *palpebralis*, or *tarsalis*) is an arrest of development, caused by interrupted growth of the cartilage and bone at the *epicanthus*.

which is attached with a more or less extensive base on the tarsal surface near the palpebral margin, and is undermined to such an extent that a sound can be inserted more or less completely between the "pseudo-membrane" and the palpebral conjunctiva. It is usually regarded as a deformity, but I have repeatedly observed this membranous duplication as a sequel to croupous conjunctivitis. Then the fold being swollen extruded like a sac, and coming in contact with the abraded tarsal conjunctiva near the palpebral margin an agglutination and adhesion resulted. In a few other cases there had been too energetic application of silver nitrate to the blennorrhœic conjunctiva. Blennorrhœa had likewise been present in Campbell's case. Rudimentary cases "in which nothing but fine lines or small dots, interpreted as cartilaginous islands, marked the beginning of a former fold," probably can be explained as non-congenital or of artificial formation (Schapringner).

Treatment.—Removal of the duplication may become necessary, especially if complicated by entropion. These cases are successfully treated by dividing the bridge-like adhesion of the transition fold to the palpebral conjunctiva at the palpebral margin, and uniting the freshened margins by several vertical lateral sutures. This also reestablishes the upper wall of the conjunctival sac which had previously been absent or completely lost.

2. CONGENITAL ANOMALIES OF THE LACHRYMAL DUCTS

The following conditions are observed: atresia of the puncta lacrimalia, the canaliculi being either present or normally permeable; absence of all or some puncta lacrimalia in the presence or absence of the canaliculi; fissure-like apertures of the puncta lacrimalia; congenital fistulæ of the lachrymal sac and simultaneous deformity of the nasal half of the same side. The congenital slit-like shape of the lower canaliculum with intact puncta, however, is often deceptive, the upper line of demarcation being formed by a thin membrane, while the palpebral margin at that place forms a groove. Lachrymal fistulæ may perhaps be explained by delayed closing off of the canal, the solid epithelial band which is the first rudiment of the lachrymal sac then acting as an obstruction. Supernumerary canaliculi may be considered as due to abnormal branching off of the canal. A fistula may be caused by an intra-uterine infectious dacryocystitis.

A peculiar congenital anomaly (persistent patency of the lachrymal groove) is the subject of a communication of Wernecke, of Odessa, to E. v. Hippel. He says: "A deep aperture, invested with mucous membrane, takes the place of the lachrymal sac. There are no puncta lacrimalia. The lids terminate in folds at the inner canthus, which are continued in the groove referred to. The aperture leads into the nasolachrymal duct, which is of normal direction and width and through which

the patient is able to breathe and exhale the smoke of a cigarette, when the mouth and nose are closed."

Suppuration of the lachrymal sac in the new-born (membranous atresia) is due to retention of the lachrymal contents and occurs oftener than was formerly supposed. It is nearly always caused by congenital occlusion of the lachrymal sac. The difficulty may be due sometimes to a retardation of the permeability through the solid rudiment of the sac, which should have occurred in the seventh month—the separating fetal tissue layer at the outlet into the nose disappearing too late or not at all, or, again, the diameter may be constricted by annular folds of the mucous membrane, or the lower end of the passage occluded by pressure of the lower turbinated bone, or stenosis may be caused by injuries to the bone during an instrumental delivery of the child. According to Mayou, the canal remains closed and there will be stagnation of the secretion if the detritus filling the canal is not aspirated soon after birth. This is because of the unusual narrowness of the lower portion of the canal or to pressure on it caused by a deviation of the lower turbinated bone. Under such circumstances, the lachrymal secretion formed in the waking state tends to find its way out toward the eye.

The affection, which usually commences a few days after birth with a scanty mucopurulent secretion from the conjunctival sac, is often mistaken for suppuration of the lachrymal sac, conjunctival catarrh, or even for conjunctival suppuration in the new-born, with the result that it is unsuccessfully treated for a long time with astringents or caustics. Usually, however, the conjunctival sac as well as the nose is normal. But pressure upon the lachrymal sac causes evacuation of a secretion which is thick at first and later becomes thinner.

Sometimes it will take weeks for large quantities of secretion to collect. This, as well as the fact that the lachrymal sac in the new-born occupies only part of the groove and is still able to expand upward to a considerable extent, before a swelling can be noticed externally, explains why it requires strong pressure upon the lachrymal sac to produce evacuation of the secretion.

If the stenosis persists for a long time, the consequence may be considerable bulging of the sac. This, however, will rapidly disappear after permeability has been established.

Differential Diagnosis.—Stenosis is distinguished from conjunctival blennorrhœa by the fact that in the latter the space between the lids is often quite full of mucus and pus, day and night. On the other hand, this secretion is arrested during sleep when stenosis exists. The swelling of the lids which develops in gonorrhœal conjunctivitis, as well as the complications of the cornea, are absent in stenosis. Greater expansion of the lachrymal sac might possibly simulate phlegmonous dacryocystitis.

Infection of the conjunctiva by the lachrymal sac is of less frequent occurrence, though a secondary infection of the lachrymal sac by inflammation of the nose and its accessory sinuses or from the conjunctiva by means of gonococci is more common. Tuberculous or gummatous processes in the vicinity of the lachrymal sac may cause similar symptoms. Lachrymal abscess may be simulated by a perforating outward of osteomyelitis of the superior maxilla due to suppuration of the dental follicles.

Under these circumstances the formation of a lachrymal fistula may render surgical interference necessary, this consisting in excision of the fistula and union of the resulting wound by sutures.

Such a procedure is less called for in bilateral than unilateral fistulæ, the former being usually situated symmetrically on both sides, a few millimetres below the ligament of the internal canthus. They are easily overlooked, because the opening is usually so fine that only the finest cannulæ and sounds can pass it.

Treatment.—Cleanliness of the eye. Whatever pressure used upon the wall should be light at first and gradually increased in strength. Any digital massage of the lachrymal sac should be outward and made several times a day for a shorter or longer period. If the puncta lacrimalia are closed, these manipulations will usually effect a permanent cure by bursting the occluding membrane at the lower end of the naso-lachrymal duct. Sometimes it happens that the procedure is accompanied by the evacuation of a copious purulent secretion through the corresponding nostril. If this methodical compression is of no avail, or if for any reason conservative treatment cannot be carried out, removal of the obstruction should be considered by careful use of the probe without incising the canaliculus. Thick sounds are used for this purpose. The best are Bowman's Nos. 3 or 4, which taper to about the size of Bowman No. 1 at the end (Plate XXI, Fig. 5). This procedure, however, should be left to an experienced ophthalmologist.

Coppez reports a case where the affection disappeared after a mid-wife had caused the secretion to be blown through the nose. In an untreated case it persisted for three years and nine months.

It is doubtful whether a certain case of croupous purulent conjunctivitis should also be included in this category. In that instance the lachrymal suppuration appeared as a complication, and the conjunctivitis did not disappear until the naso-lachrymal sac had been cleansed by careful treatment with a probe.

3. CONGENITAL ANOMALIES OF THE CONJUNCTIVA

Angiomata of the bulbar conjunctiva are very rare. They start ordinarily from the plica semilunaris or the caruncle, and do not grow until after childhood.

According to Fehr and Seefelder, the influence of pregnancy in producing them is undeniable.

Progressive, non-pigmented nævi of the conjunctiva are quite as rare. They are usually located in the immediate vicinity of the cornea and characterized by a yellowish-red discoloration, slightly transparent in parts, and a rather smooth, slightly undulated surface. The growth should be removed, owing to the possibility of its spreading to the cornea. Snell caused more fully developed nævi to disappear by treatment with ethylene sodium. Electrolysis would doubtless also be serviceable.

For pigmented nævi, see p. 40.

Congenital epithelial xerosis of the conjunctiva is apparently very rare. It has the appearance of the ordinary xerosis of the conjunctiva and, according to Agricola, occurs only unilaterally in a circumscribed region of the conjunctiva, where it remains stationary for life. As in Agricola's cases, the nævi observed by Seefelder were located in the region of the external canthus. Anatomical examinations made by Agricola showed an epidermoid consistency of the epithelium in the xerotic area.

Larger growths should be removed, especially if they interfere with the lachrymal secretion or produce any catarrhal irritation.

An abnormal development has been observed of the semilunar fold; also an absence of the caruncle, a hypertrophy and a hypertrophy of this, epidermal layers, partial duplications of the caruncle, and distinct supernumerary caruncles.

The glandular malformation described by Baquis was found clinically to be a change of the anterior temporal portion of the globe of one eye. They were tumor-like growths varying in size from a bean to a grape, of reddish-yellow color, soft glandular consistency with a verrucous surface, and covered by the bulbar conjunctiva. In the cases of Baquis and Falchi, the immediate cause of the deformity was obscure. These assumed the shape of a sinewy, white membrane and extended from one cul-de-sac to the other, even covering the temporal half of the cornea. As such growths usually cause considerable disfigurement, they nearly always come to operation. Their anatomical examination has shown glandular proliferation, embedded in a vascular and nervous tissue, consisting of connective tissue, fat, and muscles. In Baquis's case there were cartilaginous deposits and an approximation to dermoid formation.

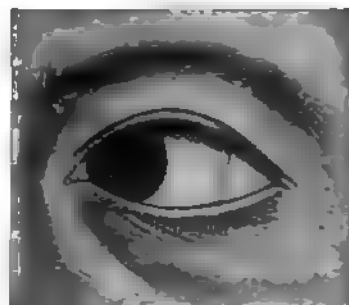
Congenital dermoids and lipodermoids of the conjunctiva often occur with other anomalies of the eye and other parts of the body. They are most frequently situated on the inferior external corneoscleral border—rarely elsewhere (Plate I, Fig. 2). This semiglobular eminence is firmly sessile upon the cornea and usually about the size of a split pea.

Hairs are sometimes found on its surface. Removal may be indicated, if it produces irritation or reduces the visual acuity by spreading over the cornea. But only the part protruding above the level of the cornea should be taken off. The resulting wound with any loss of scleral tissue may, if necessary, be covered by conjunctiva. Dermoids of the caruncle, the semilunar fold, or in the region of the transition folds are very rare. They occur isolated in the region of the inner canthus, usually between the tendons of the superior rectus and external rectus. Being sharply defined like a tumor, they project more or less above the adjacent conjunctiva, and, as they increase in size, may advance toward, or even encroach upon, the cornea. These masses also should not be cut out deeper down than the cul-de-sac in order to obviate any escape of the orbital fat. The removal of lipodermoids of the inner canthus is easier, especially as the growth is often encapsulated.

Congenital teratoid osteoma of the bulbar conjunctiva is very rarely observed in eyes otherwise perfectly healthy. It is superficially situated and therefore easily excised. This is also true of the lipodermoid variety which thus far has been observed principally in females. It has been designated by Ewetzky as the semilunar or dermoid fold, according to the character of the conjunctival anomaly. It has been regarded as a conjunctival proliferation of the palpebral conjunctiva (von Ammon), also as a supplementary or fourth lid (DuBois, Fans), also as an ectropion of the conjunctiva (Fron Müller), and as a rudimentary lid (Larcher) (Fig. 3).

It consists of a thin, flat fold situated always at the outer side of the globe. Its thickened and reddened margin lies toward the cornea, whose outer half is embraced by this semilunar margin, which does not, however, touch the cornea. The distance of the fold from the latter varies in different cases, and also changes with the position of the eye. Behind the free part of the margin there is a furrow, whose depth again depends upon the position of the globe. The upper and lower ends of the concave margin blend with the corresponding transition folds, approaching the vertical meridian of the cornea, and thereby determining the semilunar shape of the free margin. The outer part of the fold is imperceptibly lost behind the canthus. The outward appearance of this fold resembles the plica semilunaris of the inner canthus, except that it is somewhat thicker and yellowish in color. It is not adhesive to the sclera, upon which it can be easily moved, is of smooth surface and soft consistency. In some cases hairs are noticeable, while in others

FIG. 3.



Ewetzky's fold.

they are entirely absent, or are revealed only by anatomical examination. In one case a dermoid situated upon the fold extruded outside the lid fissure.

4. CONGENITAL ANOMALIES OF THE CORNEA

Congenital Opacities and Staphyloma of the Cornea.—Aside from diffuse opacities with vascularization of the cornea, which occur in microphthalmos and represent a pure arrest of development, opacities of the cornea are caused by injuries received at birth. The pressure of the forceps may tear Descemet's membrane, with resulting striated opacities in the lower strata of the corneal substance; or there may be opacities caused by infection at birth. Some of the varieties on record are an annular concentric yellow opacity running to the limbus as the result of fetal endogenous uveokeratitis of syphilitic origin; a unilateral cloudy, bluish-white corneal opacity in a hydrocephalic infant, carried to full time; and a diffuse, bluish-white opacity of the lower strata of the cornea, traversed by numerous superficial vessels, in an infant which soon afterward was attacked by a skin affection having a suspicious resemblance to pemphigus.

Tongue-shaped marginal opacities have also been observed; parenchymatous opacities at various places, which a fluorescein stain showed to be an endothelial affection; congenital ectatic leucoma; adherent and non-adherent total staphyloma of the cornea. Bilateral opacity and staphyloma are strikingly frequent.

Seefelder's investigations have shown that there may be inflammatory corneal processes during intra-uterine life, leading to disintegration of the tissue and corneal opacity.

Imperfect development of the central portion of the cornea may produce keratoconus with microcornea, cataract, or lead to the retention of remnants of the pupillary membrane, congenital retinitis pigmentosa, etc. The hereditary form, which is less frequent, has also been attributed to this cause. Irregular corneal astigmatism, without any visible precedent affection of the cornea, is also referred to some earlier form of keratitis. Chailloux refers to simultaneous occurrence of Basedow's disease, Wicherkiewicz to that of chlorosis.

Peters has described an opacity whose origin is as yet unexplained. It may be congenital or a family affection; it is nearly always bilateral, grayish-white, parenchymatous, disc-like, and lies in the middle of the cornea. There may be a deeper involvement of tissue extending even to the membrane of Descemet. This anomaly is accompanied by such other changes as hypertrophy of the globes, partial or complete absence of Schlemm's plexus, persistence of the fetal ligamentum pectinatum, typical coloboma of the iris, and embryotoxon—an abnormal extension of the episcleral tissue over the limbus in the shape of an annular opac-

ity of the corneal margin, especially pronounced superiorly and inferiorly (see Plate I, Fig. 2). This opacity may protrude like a keratoconus, the entire cornea may be enlarged and the intra-ocular pressure raised. It either clears up from the margin and completely disappears, or there remains a slightly turbid or enlarged cornea, with or without opacity. In a few cases the iris or the pupillary membrane was adherent to the margin of the opacity in the shape of a ring. There are several cases on record of permanent increase of tension, abnormal depth of the anterior chamber, and excavation of the entrance of the optic nerve, presenting the picture of hydrophthalmos.

Peters observed congenital deformity of the Descemet's membrane in one eye and congenital staphyloma in the other.

In a case which I observed, and which is described by Fr. Rückert, the normal border between the cornea and sclera was occupied by an opacity so that in a rather large portion of the corneal layers there was scar-like tissue so closely resembling scleral tissue that in some parts it could not be distinguished histologically from it. Moreover, a large part of the changed corneal area was peripherally adherent to the iris.

Dermoids and lipodermoids, often covered with fine downy hairs, are white, yellowish-white, or yellow, and have been attributed to adhesions between the globe and the amnion, or to metaplasia of the cornea. They sometimes occur in groups. Usually they are located at the outer or lower outer part of the sclerocorneal border as flat, semiglobular, or lentiform, tumor-like eminences of various sizes (Plate I, Fig. 2). Epibulbar and circumbulbar dermoids are very rare. They are sessile, on the middle of the cornea, or occupy the entire cornea and the adjacent parts of the sclera, enveloping the globe. When a microscopic examination had been made it showed a high degree of microphthalmos. A thinned pseudocornea was also found, and, adjacent to it, the iris, ciliary body, and the lens. Schlemm's canal has also appeared to be obliterated with posterior synechiæ and contraction of the lens or cataract. In other cases the lens was absent, or in front of the anterior aperture of the bulb, within the dermoid. Dermoids are sometimes accompanied by other anomalies of development, such as lid coloboma, to which reference has already been made. In that case the dermoid was located at the place corresponding to the lid defect. Sometimes there are concomitant corectopia, microphthalmos, uveal coloboma, and skin appendages.

The lattice-like opacity of the cornea, which has been observed as early as the fifth year, is often hereditary. It consists in a change in the corneal surface and substance proper, in the shape of numerous white spots and dots, which cause a gradual diminution of vision.

5: CONGENITAL ANOMALIES OF THE UVEAL TRACT

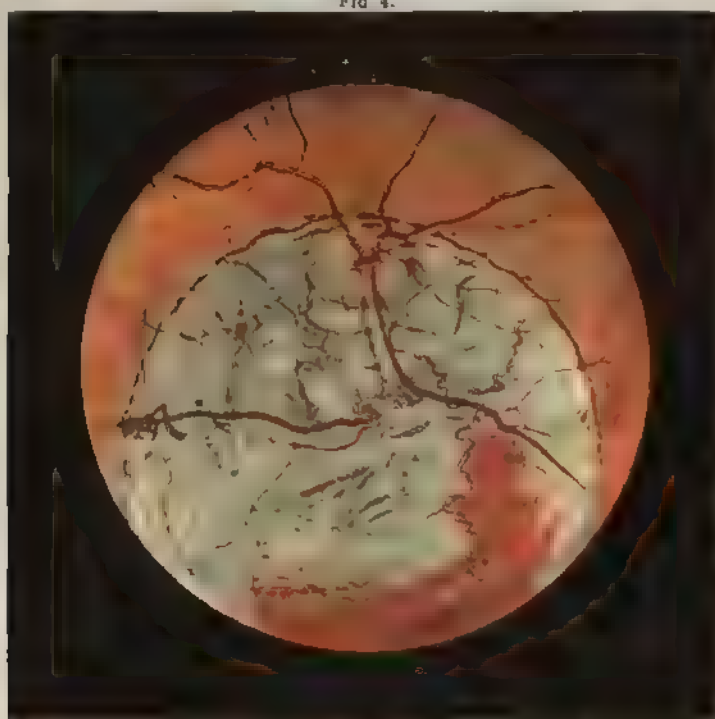
Typical coloboma of the iris is caused by delay in the closing of the fetal lid fissure of the margin of the optic cup. It is situated downward, or downward and inward. It usually occurs with other colobomas, as, for instance, coloboma of the chorioid, of the ciliary body, of the zone of Zinn, of the lens, vitreous, retina, optic nerve, or of the internal part of the sclera. Microphthalmos, remnants of the pupillary membrane, or other anomalies of development, such, for example, as dermoid and lipodermoid of the conjunctiva, verrucæ of the lid, coloboma of the lid, anomalies of the mouth, ear, and coccyx, have been observed as concomitants of coloboma of the iris. The shape of this is usually like a pear, or egg, or Gothic arch, gradually tapering from the pupillary to the corneoscleral margin (Plate I, Fig. 3). In some cases it extends to the ciliary margin; in others nothing but a peripheral fringe of tissue is left. A coloboma in shape like a keyhole also occurs, though rarely. Or it may be limited to a mere notch in the pupillary margin, the anterior layers being either missing or imperfectly developed, or the growth may be bridged over by a trabecular tissue at the small circle of the iris (bridge coloboma).

The following points should be noted in the differential diagnosis: In typical coloboma of the iris the remaining part of the iris is correctly developed, while, on the other hand, in irideremia or aniridia there is total absence of the iris. The latter is caused by an arrest of development ("diminished differentiation").

Furthermore, atypical coloboma occurs at other points on the circle of the iris, and consists of a mere displacement of the pupil (corectopia), and, in spite of the narrowness of the iris, its sphincter is still present. In typical coloboma the sphincter is absent, even though a short stump of iris tissue has been demonstrated anatomically. The pupillary sphincter is continued into the margins of a true congenital coloboma of the iris, while in an acquired coloboma of the iris—traumatic or operative—it terminates abruptly at the two places where the crura of the coloboma and the pupillary margin meet. In typical coloboma the entire pupil is often displaced downward, and the small iris circle tapers toward the lower part of the coloboma until it completely disappears. The furrows in the iris due to contraction occur only in its upper segment. The cornea likewise often tapers downward, assuming the shape of a pear, and if the coloboma continues into the ciliary body, the sclera is flattened downward by the corneal margin. There is no scleral or corneal scar in congenital coloboma of the iris, while it is never absent in the acquired form.

Coloboma of the ciliary body has failed thus far to furnish any definite, clinically demonstrable signs, because even the invisibility of

FIG. 4.



Coloboma of the choroid (After Ed. v. Jaeger.)

FIG. 5.



Coloboma of the optic disc

ciliary processes in the area of the coloboma of the iris is no proof of its non-existence. But perhaps ophthalmodiaphanoscopy (see p. 20) is destined to throw some light on this subject.

Coloboma of the pigment epithelium and of the chorioid (Fig. 4) is characterized by a round, longitudinally oval, or irregular space which, as seen with the ophthalmoscope, extends downward from the optic nerve, with vessels running over the glistening, white internal surface of the sclera. The vessels come partly from the neighboring sclera and retina, less often from the chorioid itself, and partly from the posterior ciliary vessels.

The margin of the coloboma often contains black pigment, but the rest is only slightly pigmented, if at all. There is great variety in the shape, extent, and line of demarcation of the coloboma. The retina is often present to a greater or less extent—though not in normal conditions. The sclera, too, is sometimes normal, sometimes thin and bulging, and subdivided into scleral crests.

The coloboma may begin immediately at the upper margin of the optic disc, or its area may be restricted to a circumscribed part of the optic nerve (Fig. 5). The so-called downward conus (Plate XIII, Fig. 1) may be so explained by the existence of a distinct bulging of the globe.

Central visual acuity may be normal in small colobomas, but in larger ones it is impaired somewhat by the resulting myopia. This holds good also for the field of vision, which is affected by the condition of the retina in the area of the coloboma.

In making a prognosis it is important to observe that a dragging of the retina at the margin of the coloboma may later cause detachment, gradually becoming complete and leading to complicated, inoperable cataract.

While the origin of typical coloboma has been established as being due to failure of the fetal optic fissure to close, the cause of the much rarer atypical coloboma, which occurs in various other areas of the iris, chorioid, ciliary body, and lens, has not yet been fully cleared up. This is true also of coloboma of the globe, which is accompanied by facial clefts and anomalies of the nose and lids. It is possible that the atypical coloboma of the fundus is in part attributable to heterogeneous pathological changes of the chorioid, such as interpartum hemorrhages, etc. This applies particularly to the so-called macular coloboma (Plate XIX) which occurs bilaterally more often than unilaterally, and appears ophthalmoscopically as a defect of the pigment epithelium and chorioid. It is sometimes ectatic, sometimes in the level of the fundus, and is sharply defined, circular or oval, white or variously pigmented. Under certain conditions it may not even cause any noteworthy impairment of vision.

Microphthalmos is to be attributed, like typical coloboma, to abnormal persistence of the mesoderm in the fetal optic fissure, and to imperfect development of the vitreous, which in turn is dependent on

abnormal development of mesoderm connective tissue in the deeper portions of the eye, and to adhesions of this tissue to the retina and lens. Microphthalmos often occurs with orbital cysts, coloboma, and other malformations, such as hydrophthalmos, congenital cataract, persistent hyaloid artery, cysts of the lower lid, epibulbar dermoids, facial clefts, polysyndactylism, and albinism.

The folds resulting from a duplication of the retina, and the adhesion of these folds among themselves as well as to the mesoderm and the lens, together with cell proliferations such as exist in glioma, were sufficient in Helfreich's case to lead to erroneous diagnosis of glioma. The following important congenital changes have been found in microphthalmos without coloboma: persistent pupillary membrane, irideremia, atrophic spots in the retina and chorioid, abnormal persistence of the hyaloid artery, opacities in the vitreous with detachment or rupture of the retina.

The so-called pure microphthalmos is simply an unusual smallness of the globe, without other ocular changes. There is often considerable hypermetropia, up to 20 D and more. The deep position of the globe, usually with normal development of the orbit, is noteworthy. At the same time, the central acuity of vision is usually reduced, even after correction. On the other hand, Martin found normal or nearly normal visual acuity in thirteen members of a microphthalmic family, distributed through three generations.

It is noteworthy that, owing to the imperfection of these eyes, enucleation has repeatedly become necessary on account of glaucoma, and that they are often attacked by retinitis punctata albescens, pigment degeneration of the retina, and insidious chronic iridocyclitis.

Congenital total or partial absence of the iris (aniridia or irideremia) is attributable to arrested development of the entire rudimentary iris structure. It has been found to be hereditary more frequently than any other deformity of the eye. With absence of the iris are often associated an abnormally small lens, anterior or posterior polar cataract, imperfect development of the zonula, and congenital or acquired luxation of the lens. Nystagmus and lowered vision are present because of the imperfect development of the macular region or total absence of the fovea centralis. Total aniridia in one eye, and large atypical coloboma in the other, have also been clinically described. It is possible that a large, widely gaping coloboma of the iris may closely resemble a so-called partial irideremia. On post-mortem examination Seefelder found a congenital absence of the fovea centralis in an eye with congenital aniridia.

Upon examination no iris muscle-fibres could be demonstrated with any degree of certainty. Total absence of the iris is rare. In most cases more or less extensive remnants of the iris can be demonstrated clinically. Aniridia strongly predisposes to glaucoma. Twenty-six cases of secondary

glaucoma or hydrophthalmos have been found in eighty-two such cases. That is explained by the fact that the drainage angle is partly or completely occluded by so-called fetal trabeculæ. This condition seriously disturbs the normal relation of the venous sinus and the anterior chamber.

Other rare anomalies of the iris in symmetrical places which unquestionably correspond to the palpebral fissures are complete obliteration of the pupillary portion, due to reduction, splitting off and interlacing of trabeculæ radiating to the pupillary margin (W. Lohmann); complete absence of trabeculæ in the pupillary and ciliary portions of the iris or induration at the site of the fetal fissure along with circumscribed pigment proliferation (Ph. Arnold). Other rare anomalies are circumscribed absence of or imperfect pigmentation in any portion of the iris pigment layer which corresponds in direction, form, etc., of the pupillary margin to a typical iris coloboma with multiple indentations (Gilbert); also more or less extensive defects in the stroma of the iris have been observed, the presence of which in the stroma led to dehiscences in the pigment epithelium. According to Thyé and Engebrecht, these cases have also been observed as hereditary. In many cases the anterior chamber is filled with a net of fine gray threads, connecting the iris with the posterior corneal surface. Congenital cat's eye (Fehr) and a wall-like elevation of a small circle of the iris which persisted when the pupil was artificially dilated (Fleischer) have also been described. Congenital anterior and posterior synechiæ, dependent upon development, have been described by Peters and Nieden.

Scleral translumination of the eyes may be of considerable value in the establishment of congenital anomalies of the iris, and for the elucidation of many details in intra-ocular coloboma.

Thus, Langenhan, using Hertzell's ophthalmodiaphanoscope, repeatedly found in normal eyes, especially those of children, fissure-like defects in the section of the iris corresponding to the fetal fissure of that membrane. This rather rudimentary form of coloboma was restricted to the pigment layer of the iris. Such defects were sometimes confined to the peripheral portions of the iris in the vicinity of the lower median line, and sometimes they invaded the entire width of the iris, usually tapering from the periphery toward the pupillary margin. There were various transitional forms between these two, sometimes mere suggestions of defects. Langenhan thinks that there is a parallel between the absence of pigment just described, and the congenital downward partial leucosis retinæ (Pause, Manz) which Manz regarded as a minimum degree of fissure formation.

The clinical and anatomical picture of serous cysts of the iris does not seem to differ from that of traumatic cysts. Their origin requires further elucidation, since up to the present the only cysts which have

been examined histologically are those which have been already removed by operation, no attention having been paid to their surroundings.

The cause of polycoria is not yet clear. We only know that even in a normal, or especially in an excentrically placed, pupil there are frequently imperfections here and there in the iris, which are radially or irregularly arranged.

Ectopia Pupillæ (Corectopia).—Slight deviations in the position of the pupils are very frequent (Plate IX, Fig. 5). The exaggerated excentric position of the pupil, which is attributed to various mechanical impediments in the development of the rudimentary iris, is often associated with luxation of the lens. When the two conditions coexist the displacement of the lens is usually the reverse of that of the pupil. Persisting pupillary membrane, epibulbar dermoid, coloboma of the lid, microphthalmos, albinism, and buphthalmos are also mentioned as complications, as well as discoloration and irregular pigmentation of the iris and convergence of the radial fibres toward the narrow parts of the iris. In order to distinguish between coloboma of the iris and corectopia, it is important to determine whether any portion of the iris is movable, and if so which. The muscular structure in corectopia is still present in the narrowest part of the iris, whereas there are no iris fibres whatever at the apex of the coloboma.

Congenital eversion of the pigment layer of the iris (congenital ectropium uvulæ) means that the pigmented layer of the iris was reflected over its anterior surface. It usually appears in the shape of bilateral excrescences, commonly situated at the superior pupillary margin. They look like the so-called (physiological) uvæ of the horse, sheep, goat, or pig. A rare form is the ruffle ("*halskrause*," Hirschberg), this being a neatly folded fringe protruding from the pupillary plane of the pupillary margin (usually the upper part). Just as rare is the apron ("*schürze*," Hirschberg) which, running around the pupillary margin, is reflected onto the anterior surface of the iris. Upon superficial inspection this may simulate a coloboma of the iris. A. Brückner found the mesodermal tissue of the iris adjacent to the ectropion much denser and pigmented, after the manner of a circumscribed nævus.

There are also mixed forms, between ectropion and proliferations of the pupillary margin.

Gallenga attributed the origin of these anomalies to prolonged existence of the annular sinus, and to a modification of the usual relations between the pupillary membrane and the extreme pupillary portion of the pars iridica retinæ. Their origin may also have some connection with traces of the pupillary membrane.

Seefelder found an ectropion of the pupillary margin—retroversion—and E. Enslin saw an entropion of the iris in a young baby.

6. CONGENITAL CHANGES OF THE SCLERA

Thinning of the sclera in the new-born persists only in comparatively few cases. Peters observed in several members of one family a blue discoloration of the globe which had been inherited for four generations, and was caused by the pigment shining through an abnormally thin sclera. Buchanan observed this associated with a keratoconus. Circumscribed thinning of the sclera at the limbus or in the region of the palpebral fissure occurs alone or in conjunction with congenital corneal opacities (Percival, Hay, Seefelder).

Congenital scleral cysts at the corneoscleral border rarely occur. They have been attributed by Villard to an invagination of the conjunctival or corneal epithelium at a very early stage of eye development.

Congenital or inherited abnormal thinning of the sclera at the posterior section of the globe may predispose to myopia.

Treatment.—It is not advisable to attempt the removal of an entire growth on the sclera, since its posterior wall is formed by the innermost layers of the abnormally thin sclera. The conjunctiva having been dissected free, it will be sufficient to cut off the anterior cystic wall and unite the conjunctiva over the inner wall.

Peripapillary distention of the sclera in the region of the entrance of the optic nerve is etiologically quite obscure. In these cases the vicinity of the optic nerve is deeply excavated posteriorly over rather a large area. The margins of the excavation are sharp, and so steep that the vessels arising from the normal position of the optic disc partly disappear at the margin. The optic nerve enters either in the middle or toward the periphery of the ectasia, and in the latter case is only partially visible. The base of the ectasia is usually somewhat lighter than the fundus generally, but still has a reddish color, showing that the pigment epithelium and chorioid are present. Around the excavation, on the level of the rest of the fundus, there is a ring of atrophied chorioid with pigment proliferation, as it exists at the border of chorioid coloboma. There are no marked irregularities of the base of the excavation. The difference of refraction between the latter and the rest of the fundus is quite marked in all cases, varying between 12 and 16 D. The visual acuity of these eyes is in all cases somewhat less than normal, but still serviceable, often 6/8 to 6/36.

In Zade's case the optic nerve, which entered in a deep excavation, presented considerable changes; the margins were blurred and the anomalies of the vascular arrangement, etc., were present. It was remarkable that the vessels, in spite of the depth of the excavation whence they arose, did not show distinct kinking, but seemed to lie superficially.

7. CONGENITAL ANOMALIES OF THE LENS

Lenticonus posterior is a conical bulging of the posterior lenticular surface. As a rule the centre of the posterior surface is affected, although sometimes the cone is eccentrically placed. It is frequently associated with disturbances of involution of the hyaloid artery. The capsule may be thin, but otherwise intact. Rupture of the posterior capsule, however, has often been found with extravasation of lenticular substance into the vitreous. Displacement of the lenticular nucleus backward often occurs. Posterior polar cataract is present in many cases. Ectasia of the anterior surface of the lens (*lenticonus anterior*) is rare, and *lenticlobus anterior* rarer still.

Ectopia, or displacement of the lens, which is usually symmetric, is often hereditary (Plate IX, Figs. 5 and 6). It may occur alone or in conjunction with coloboma of the iris, etc., *corectopia*, *aniridia*, and *microphthalmos*, and is due to a defect or irregular development of the suspensory ligament of the lens. It is usually bilateral. The lens is usually displaced upward and sometimes abnormally small (*microphakia*); it is more often clear than opaque.

Treatment.—Concave glasses sometimes improve the impaired visual acuity in *lenticonus*. In *ectopia*, concave glasses will compensate for the myopia caused by the increased curvature of the lens, while strong convex glasses are indicated for the *aphakia*. If correcting glasses, however, fail to give serviceable improvement of the vision, or if disturbing monocular double vision persists, operation should be resorted to. This is particularly indicated if gradual loosening of the zonula causes displacement of the lens, thus predisposing the eye to secondary glaucoma. This is especially important if the patient's work necessitates much eye strain. Functional tests with normal and dilated pupils will show whether iridectomy, sphincterotomy, or sphincterectomy is the indicated operation, or whether the lenticular contents had better be early evacuated by discission. In order to prevent, among other mishaps, a prolapse of the vitreous due to the defect of the zonula, general and local anæsthesia are absolutely necessary to ensure good results, such as I have repeatedly obtained.

Aside from opacity at the posterior pole of the external surface of the capsule due to residues of the vascular capsule, and aside from the fibrous pseudo-lens resulting from the tunica vasculosa, there are congenital uni- and bilateral changes of the lens to be considered, which occur most frequently as inherited affections. These changes consist in luxation of the lens, which occurs sooner or later, and in the various forms of cataract—nuclear, fusiform, anterior or posterior polar, laminated or total cataract—which are often complicated with disturbances

of the eye due to imperfect development. Such complications are microphthalmos, coloboma of the lens or iris, aniridia, persisting tunica vasculosa, persisting pupillary membrane, and medullated nerve-fibres. General complications are: distinct signs of rhachitis, telangiectasia of the facial and general skin, pachydermia, nævus ichthyosiformis, atrophy of the eyebrows, orbital cyst, micrognathia, microcephaly, plagiocephaly. In some cases the thyroid seemed to be absent. It is not yet definitely established whether the various forms of cataract, which occur alone or associated with other complications, are inherited arrests of development and deformities, or whether they are the results of pathological changes possibly due to intra-uterine affections of the eye. So far as laminated cataract is concerned, there is a probability of both intra-uterine and post-natal origin. Microphthalmos, which is usually present, the demonstrable injuries to the posterior lenticular capsule through mesoderm cords, rupture of the capsule, retarded ligation of the germinal vesicle, hyperdevelopment and insufficient involution of the tunica vasculosa, and the presence of other lenticular opacities, point to intra-uterine origin of laminated cataract and the related forms with punctiform opacities. These opacities may be scattered through the entire lenticular substance, or collected about the equator of the lens (*cataracta punctata*). These abnormal conditions may be associated with either opacities in the vicinity of the anterior pole of the lens which resemble, when fully developed, a small bird's feather (*star cataract*) or with elliptic opacities of the posterior cortical substance (*central and fusiform cataracts*). Thus, v. Szily was able to trace certain cataract forms to scattered cell groups of atypical rudimentary arrangement, while in a congenital total cataract Gilbert demonstrated an extensive rupture of the posterior capsule. At the spot where this rupture occurred there was a residue of the tunica vasculosa.

Peters and Nettleship state that inherited laminated cataract may develop either in fetal life or in the first months after birth. Both forms have been observed in one family. According to Nettleship, this form of cataract is to be regarded as congenital if there are no deformities of the permanent upper incisors. According to v. Hippel, hereditary inferiority of the epithelial corpuscles, or tetany, may be concerned in congenital cataract.

Congenital cataract associated with heterochromia iridis congenita is no doubt due to fetal uveitis.

Laminated cataract in the course of time may become intensified. But it is sometimes so delicate that it causes no disturbance whatever, consisting anatomically of a regular zone of numerous droplets of greater refraction than the body of the lens. These probably lie between, and not in the fibres (Plate IX, Figs. 3 and 4). Such droplets are also found in the nucleus, but only in small numbers.

In central cataract the entire nucleus is infiltrated with large quantities of droplets, or there is pronounced disintegration. In these cases the nucleus often lies very far behind, adjoining the posterior capsule.

In fusiform cataract the central opacity is supplemented by tubular opacities, extending to the anterior and posterior poles.

All these forms are usually stationary. The peripheral lenticular layers adjacent to the opacity are either normal or adherent, but there are few circumscribed opacities. Complete involvement of the lens in advanced life occurs only in rare cases. Congenital total cataract (Plate IX, Fig. 1) often has close anatomical relations to laminated cataract or to central cataract. Finally, extensive liquefaction and resorption produce a so-called cataracta membranacea, and prolific lime deposits are often seen within the *débris*.

There are also large capsular cataracts of widely varying shapes which have originated in the capsule epithelium and also contain lime deposits.

The anterior polar cataract is a circumscribed capsular cataract, usually perfectly round, which occurs either alone or in conjunction with any of the other forms mentioned.

Posterior polar cataract consists of a cell proliferation inside the lenticular capsule. An opacity of the posterior portion of the anterior capsule may have the same appearance. Then it is a false posterior polar cataract, often with the remains of a capsular membrane or hyaloid artery.

Nettleship and Ogilvie have established hereditary cataract in eighteen members of a family of two hundred and eighty-eight persons, comprising seven generations ("The Coppock Cataract"). It consisted of a central opacity behind the lenticular nucleus, but in front of the posterior lenticular capsule, and was symmetrical in form. Visual acuity was only slightly affected. Usually there was no complaint except photophobia, indicated by the fact that these individuals shade their eyes with the hands, when looking closely at an object in bright light. The cases in this family have so far been found only in persons over ten years of age.

As to *treatment*, congenital fetal cataract demands early operation, when the general condition is favorable, because the removal of contracting and calcifying cataracts is much more difficult. Early removal is also of the greatest importance in order to prevent amblyopia from non-use, for obtaining better vision, for causing disappearance of the nystagmus which impairs acute vision, and for promoting mental development. Hirschberg states that these children often remain weak-minded, even if operated on for cataract early in life. I have frequently successfully operated upon infants in their first year of life, performing dissection of the capsule and evacuation of the cataract.

Discission of the capsule is not a suitable operation in so-called congenital hard nuclear cataract, which rather requires extraction. If there is a suspicion of complicated cataract following fetal, insidious iridochorioiditis—for instance, of syphilitic origin—the operation should be preceded by general treatment.

The regenerative tendency of the infantile lens is so great that even after successful removal of congenital cataract, including discission of the posterior capsule, the operation may have to be repeated several times in later years. Should this involve the danger of considerable pulling of the ciliary body, or of touching the ciliary processes which protrude into the pupillary space, it may become necessary to tear the capsule with two needles (which is not always an easy matter), or to extract it. This protruding of the ciliary processes is often due to equatorial contraction and increased density of the capsule, with the possibility of a grave intra-ocular hemorrhage.

In one of my cases considerable elongation of the processes of the entire ciliary body had taken place. After the clinical examination had established a secondary and not excessively thick cataract, under complete local anæsthesia, I opened the chamber from below and outward with a moderately broad lancet, introduced Elsberg's discission scissors, and smoothly divided the cataract with one short clip. This was without any untoward accident. The result was a perfectly black, uniformly round pupil which reacted well to light, etc. (*Münch. med. Wochenschr.*, 1901, page 49). In a similar case, R. Schweigger, employing general anæsthesia, sharply dissected the ciliary processes free from the cataract, in which he made several incisions with Schweigger's scissors. The pupil thereupon became black and was of normal size. The remnants of the ciliary processes withdrew, although not completely.

Partial Stationary Cataracts.—If the visual acuity is not sufficient for reading and writing, if accommodation is very slight, if myopia has already set in and spectacles or dilatation of the pupils by daily instillation of a 1/10 per cent. solution of atropin do not give sufficient relief, or their continued application is inadvisable, the choice of operation rests between providing a small optical pupil, evacuation of the lenticular contents by discission, or so-called lineal extraction with the lancet knife. In making an artificial pupil it is best to preserve the periphery of the iris by doing a sphincterectomy. In iridectomy, refraction and accommodation remain intact, and the iridectomized eye is always superior to the aphakic one. It is, therefore, particularly suitable in cases where the marginal parts of the lens are clear, and the test for visual acuity after dilatation of the pupil with a 1 per cent. homatropin solution shows adequate improvement. Should this test prove unfavorable, extraction is indicated in the presence of considerable opacity of the lens periphery.

Amblyopia ex anopsia is not to be apprehended so much in these cases as in total congenital cataract. There is no need, therefore, of performing evacuation as early as the first years of life, especially in unilateral, congenital partial and stationary cataracts, such as the laminated forms, etc. Should the opacity involve the poles and axis of the lens, which often causes considerable reduction of central visual acuity, I perform an upward "temporary iridectomy," provided the growth of the cataract is very slow. In such cases an iridectomy somewhat broader than the ordinary optical iridectomy may effect considerable improvement.

As the lens at birth is non-nucleated and is of a uniformly soft, limpid consistency, gradually becoming more yellow and dense, the growth of the infantile globe may be retarded by operative treatment (Wessely).

Coloboma of the lens also occurs in conjunction with ectopia, and may be solely due to incomplete development and tension of the zonula. The lenticular margin is flattened in but one place, usually at the bottom, or it may be more or less notched. In Fleischer's case two small brown cords ran from the ciliary body to the lens. Coloboma of the zonula can be recognized by the ophthalmoscope because of the absence of fibres, if there is a gap in the iris.

8. PERSISTING REMAINS OF THE TUNICA VASCULOSA LENTIS

The clinical picture of this anomaly, which occurs more frequently in one than in both eyes, is often associated with other deformities of the eye, such as microphthalmos, irideremia, coloboma of the iris, ectopia of the lens, etc.

Persisting remains of the membrana capsularis are rare. The most important is the anterior part of a persisting hyaloid artery, which varies according to size, shape, and appearance, and is deposited upon the posterior surface of the posterior lenticular capsule (Plate IX, Fig. 2). It may also be associated with posterior polar cataract and opacity of the posterior corticalis. Small as well as extensive obliterated remnants of the posterior section of the tunica vasculosa have also been observed with the hyaloid artery, their ramifications generally agreeing with those of distribution of the fetal blood-vessels in this area. The irregular development of gliotic and mesodermal tissue of the persisting membrana capsularis formed such a firm membranous deposit in several cases as to simulate glioma and pseudoglioma. Light brown dots are also found resembling those seen on the anterior capsule as rudiments of the anterior section of the tunica vasculosa. In like manner, remains of the pupillary membrane may also be present simultaneously with those of the posterior capsular membrane.

Cases in which a persisting hyaloid artery and its ramifications do not extend to the posterior capsule may perhaps be explained in part as a detachment of the capsular membrane from that posterior capsule.

As to the differential diagnosis, the remarks made in regard to the hyaloid artery hold good here. The simultaneous presence of vitreous opacities does not necessarily exclude a fetal remnant. On the other hand, Brückner observed pigment dots at the posterior lenticular surface in chronic iridocyclitis.

Treatment.—It is an open question to what extent a serious functional disturbance, such as may be caused by polar cataract of the posterior corticalis or vitreous opacities, can be benefited by treatment, if at all. Optical iridectomy, or removal of the lens by discission and evacuation, might, for example, be considered in opacities of the lens, if the other eye is irremediably blind, if a diagnostic dilatation of the pupil holds out the hope of a notable improvement of the visual acuity, and if the ophthalmoscopic examination reveals no other complications.

FIG. 6.



Persisting pupillary membrane.
(After E. v. Hippel.)

Remnants of part of the tunica vasculosa, which extends from the lenticular equator to the retroverted margin of the secondary optic vessel (membrana capsulo-pupillaris), as well as remnants of the capillary net of the equatorial region of the lens, connecting capsular and capsulo-pupillary muscles, are still rarer conditions. They have been described as white, sharply defined opacities extending from the margin to the posterior surface of the lens—also as fine threads, and

as rust-brown pigmented lines of varying shape and extent.

In coloboma of the iris, or iridodialysis, they are visible only with maximum mydriasis.

Remains of the anterior section of the tunica vasculosa and of the pupillary membrane (Fig. 6) are seen very often. Sometimes a few brown dots and threads on the anterior capsule are the only indication. If they are at all numerous, their arrangement may show the distribution of vessels of the anterior fetal tunica vasculosa. Other threads may span the pupil in a straight or oblique direction and attach themselves to the opposite part of the iris. Or a fine thread may run from the surface of the iris to a small, white, roundish capsular opacity in the middle of the pupil.

The following, however, is the more common condition: the threads, which may have the same or a lighter color than the stroma of the iris, interlace with each other. They arise in varying numbers and strength from the region of the small iris circle, and sometimes extend peripher-

ally to the ciliary part, and even to the root of the iris. In rare cases they arise from the pupillary margin. Instead of interlacing, they may also terminate singly in the pupillary region, or cover the latter. Less frequently they form a membranous, white or pigmented, irregular plate which is either centrally located and is adherent to the anterior capsule, or floats free before the lens. On the other hand, the entire pupillary membrane may be preserved, and be so dense that it gives the impression of an old exudative membrane.

The following case, observed by me and published by Ph. Arnold, is rare and atypical:

The downward and inward part of the small circle of the iris approached nearer the pupillary margin; the circle uniformly drew away like a snail toward the inner upward and the outward side. Its shape was altered downward and outward by two cone-like projections reaching into the pupillary region from the iris tissue, and between which the pupillary margin reappeared. On inspecting the surface of the iris, the lower cone-like projection looked like the protruding zig-zag line of the small iris circle. The outer and lower piece, which was broader, resembled an enlarged formation of the structures of the small iris circle, such as appear upward and outward in the pupillary part of the iris. The pigment layer of the iris was slightly everted upward and downward, and the only part completely covered with iris stroma was the one pointing inward and downward. The cone, as described, advanced when the pupil reacted. The right pupil was larger than the left, and reacted sluggishly. The refractive media were clear, and both discs showed physiological excavation and *conus temporalis*.

The differential diagnosis between this form and posterior synechiæ of the iris of extra-uterine origin is usually easy, if all the etiological and clinical circumstances are taken into consideration, because all the synechiæ are situated at the pupillary margin itself, while the threads of the persisting pupillary margin usually originate at the anterior surface of the iris.

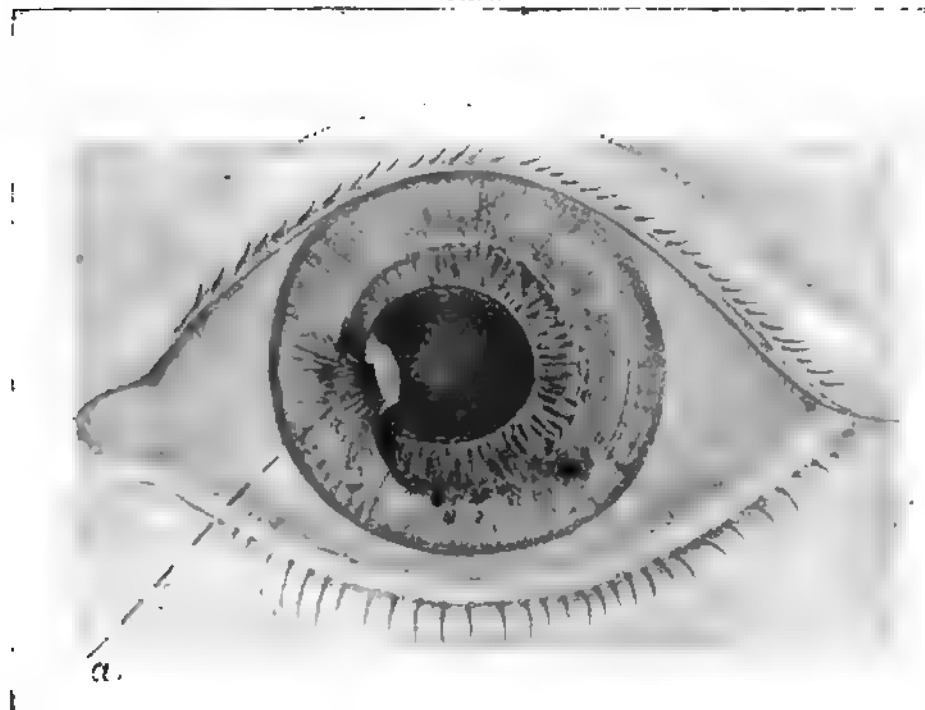
The behavior of the retroverted margin of the pigment layer of the iris, as well as that of the pupil, when artificially dilated, deserves special consideration. It is generally an indication against synechiæ if, in spite of threads which are fixed upon the lenticular capsule, the pupil remains round under maximum mydriasis. Round or star-shaped and lighter colored residues on the capsule may also be considered. The differentiation is more difficult between them and the remains of intra-uterine inflammations, especially if there are surface adhesions between the pupillary membrane and the anterior lenticular capsule.

Adherent pupillary membrane of the cornea is sometimes attributed to development arrested prior to the final formation of the anterior

chamber, to "partial failure of separation of the rudiment common to cornea and pupillary membrane," to apposition of the iris at a place where the posterior surface of the cornea has been deprived of its endothelium, and to an adhesion of the iris to the cornea, following perforation of a corneal ulcer in blennorrhœa neonatorum.

Freely movable clumps of pigment in the anterior chamber, and the brown and gray lines and spots attached to the back surface or the lowest parts of the cornea, are of the same diagnostic interest.

FIG. 7.



Hyperplasia of the infero-interior sector of the iris, and cataract of the anterior capsule (a). (After Haeberlin.)

Other congenital anomalies intimately connected with development of the persisting pupillary membrane are: irideremia, coloboma of the iris and chorioid, ectropion of the pigment layer of the iris, and corectopia; anterior central cataract, total cataract, circular cataract, deep opacities of the lens resembling laminated cataract, and dense, lenticular, whitish glistening or whitish-blue, flat, compact subcapsular opacities of the lens (Brückner).

I have observed the following anomaly which is described by Haeberlin: A coarse, compact tissue flap, comprising about the sixth part of the total circumference of the zone of the small circular iris and of the same appearance as a peripheral iris section, bridged over the pupillary margin.

This flap terminated in a strongly pigmented fringe nearly 2 mm. wide, situated on a ridge of the anterior capsule of an opaque lens (Fig. 7). Brückner considered this case a very strongly developed residue of the pupillary margin, while Haeberlin explained it as an irritation which led to hyperplasia of iris tissue in a circumscribed area of the iris surface. So far as our present knowledge of the history of teratological development permits, I am inclined to think it more probable that the starting point of this hyperplasia was a circumscribed, very early intra-uterine tear of the anterior lenticular capsule, which was followed by an anterior capsular cataract and adhesion to the fetal iris.

Other diagnostic points of importance are that abnormally persisting remains of the pupillary membrane often undergo involution after the first period of life, that they occur more frequently in strongly pigmented eyes than in those that are less so, and that the pupillary membrane may participate in a kerato-iritis.

Treatment.—Tearing the threads by alternating miosis and mydriasis may promote resorption. Hasner removed a plate resting on the anterior surface of the lens. Inasmuch as vision is rarely disturbed, unless the residues of the pupillary membrane are very extensive, the condition should not be interfered with, except when complicated by total or zonular cataract.

9. CONGENITAL ANOMALIES OF THE VITREOUS

The eye of the new-born child contains a remnant of the hyaloid artery about 1 to 1.5 mm. long, and in addition part of the residues of embryonal fibres and cells. The latter may be regarded as the anatomical foundation of the so-called *muscæ volitantes*.

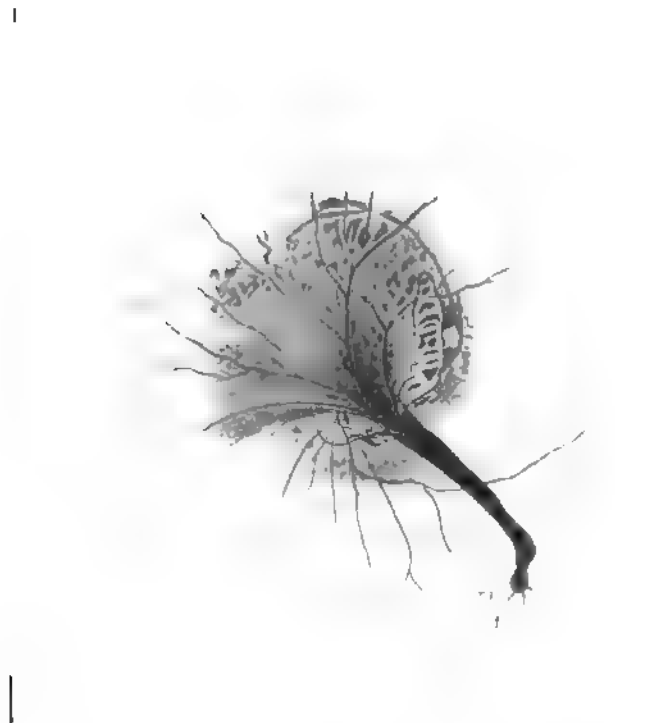
Persisting hyaloid artery has been found to occur almost without exception unilaterally in eyes that were otherwise clinically normal. The condition may also be associated with persisting mesoderm processes in the fetal fissure, along with remnants of the *tunica vasculosa lentis*, *lenticonus posterior*, persisting pupillary membrane, *coloboma*, and *microphthalmos*.

The ophthalmoscope shows a cord-like structure originating at the central artery or from one of its branches, and extending from the entrance of the optic nerve to the posterior surface of the lens. Here it is attached to the capsule. This point of attachment may be a punctiform, conical, or globular thickening from which fine opaque branching threads radiate, or in the shape of a membrane. The cortex lens at the posterior pole is often opaque. When the eye is moved, the complete structure participates, like a rope stretched between two points, or it may be like the lash of a whip in cases where the connection with the lenticular capsule is missing. Sometimes the artery does not extend to

the lens, but breaks up into fine hairs in the vitreous, while there is no connection between the posterior and anterior parts of the structure.

Persistence of the glia coat, which normally accompanies the fetal hyaloid artery, or condensation of the surrounding mesodermal tissue, presents the picture of a utricular structure with delicate transparent walls, forming in its posterior part a kind of ampulla or tent-roof, which partly or totally hides the central vessels. Kersing and I were the first to establish the clinical fact that these utricular structures may merge into net-like membranes, which are detached from the optic disc and

FIG. 8.



Persisting hyaloid artery with condensed glial coat Detached net-membrane. (After O. Eversbusch.)

drawn forward (Fig. 8). "Vascularized connective tissue" has also been observed directly behind the lens, at the end of a persisting hyaloid artery, leading to the erroneous diagnosis of glioma.

Vestiges of the vasa hyaloidea propria, which are no longer filled with blood, have been described as numerous fine hairs of regular shape, diverging in different directions in nearly straight lines toward the periphery of the vitreous and disappearing imperceptibly.

It is not always easy to decide the nature of these residues in an individual case. As a matter of fact, a congenital utricular structure has been erroneously diagnosed as an encapsulated cysticercus, and as

a new growth, indicative of glaucoma. On the other hand, I observed in the vitreous, following a serious injury to the eye, the residue of a hemorrhage with a connective-tissue scar (Klin. Monatsbl. f. Augenheilk., 1899). A hemorrhage had taken place in the middle part of the central vitreous canal, without having perforated the normal, anatomically preformed fissures and gaps of the vitreous, thus presenting an ophthalmoscopic picture extremely similar to that of persisting hyaloid artery. Numerous processes of varying length radiated in a uniform manner from the margins of a connective-tissue plate in front of the disc, and nearly in the middle of the vitreous axis. These processes seemed to be enveloped in a glistening mantle, becoming narrower in their course toward the periphery, and, tapering to a hair, terminated as sharply demarcated lines.

As to the case of Wilh. Reis, which he described as a "Congenital connective-tissue diaphragm in the vitreous of the left eye, with a large fundus coloboma, and coloboma of the macula lutea of the right eye," I am inclined to believe that this case can without hesitation be explained as a sequel to an extensive intra-ocular hemorrhage (interpartum). This is especially because analogous membranous formations often follow retinal hemorrhages, as in chronic interstitial nephritis, etc. I also think that the fundus changes designated as "coloboma of the macula lutea" have not as yet been sufficiently substantiated in the history of development.

10. CONGENITAL AND INFANTILE GLAUCOMA (HYDROPTHALMOS CONGENITUS)

The intrinsic connection of hydrophthalmos (Fig. 9)—which is sometimes hereditary—with neurofibroma of the ciliary nerves, with elephantiasis mollis of the lids, and with neurofibroma of the facial cutis and the orbita, is still unexplained. These affections are anatomically and pathologically characterized by connective-tissue proliferation at the peri- and endoneurium of large and small nerves of the skin. V. Michel found this proliferation in the form of fibroma nodules in the anterior ciliary nerves of the hydrophthalmic eye. Other similar anomalies are: nystagmus, corectopia, ectopia of the lens, coloboma, glioma, irregular development of the ears (one concha being more massively developed than the other), dental caries, and others.

The true giant cornea (megalocornea), which is very rare, and megalophthalmos, belonging to the same category, differ from hydrophthalmos or juvenile glaucoma in the fact that the eyes are normal. Corneal opacities, enlarged and occasionally oval pupil, and the consequent atrophy of the iris (which in one of my cases appeared with extensive gaps in the stroma of the anterior layer, and had even perforated parts of the iris) are all present in absolute juvenile glaucoma, but

are absent in megalocornea and megalophthalmos. Nor is there with these increase of tension, pressure excavation at the optic disc, or the usual progressive failure of vision. Megalophthalmos is often an abortive form of hydrophthalmos and has also been observed in several members of one family. Nevertheless, this enlargement of the corneal base, which Horner designated as a pure cornea globosa, deserves attention owing to the tendency of these cases to develop freely movable cataracts later in life.

Hydrophthalmos may occur in different ways. Congenital anomalies of the principal excretory ducts of the anterior ocular segment are nearly always associated with a deep anterior chamber; abnormally small, posteriorly placed, partial or complete absence of the venous spaces of

Schlemm; and imperfect development of the so-called scleral spurs, with abnormal persistence of the fetal, uveal trabeculae, has been observed in many cases. The primary condition, according to Seefelder, is impeded filtration. Inflammatory processes and vasomotor disturbances are always secondary, and important only in so far as they are intercurrent factors.

Physostigmine instillations will easily decide dubious cases. At least, whether after its use a corneal opacity disappears for a short time or considerably decreases, or whether the enlarged



FIG. 9.
Congenital hydrophthalmos of the left eye.

eye shows opaque spots of the cornea with what Haab has described as "peculiar ribbon-like striæ deep in the corneal substance, caused by tears in Descemet's membrane, in which they pursue a peculiar, tortuous course," he observed that the centre is less opaque than the parallel margins around it. The latter, when inspected through a loup, are found to be formed by lines resembling glass threads.

In congenital glaucoma the earlier treatment begins, the more favorable is the prognosis. In many cases the disease can be arrested, but the initial stage is often overlooked, and medical aid is not invoked until too late.

The *treatment* consists of instillations of the principal palliative miotics, continued for years: Pilocarpin. mur., 0.5 to 2 per cent.; physostigmin. salic., 0.25 to 1 per cent., and morphin. mur., 0.01 to 0.05 per cent.

Pilocarpine has a weaker, but more uniform, effect than physostigmine, although this varies with the individual. Pilocarpine requires much larger doses than physostigmine to produce miosis, spasms of accommodation and reduction of the intra-ocular pressure. Unlike physostigmine, pilocarpine produces no latent increased excitability after the visible effect has worn off. The pupil again becomes larger than normal, accommodation difficult, and the near point is farther away, as before.

Congenital glaucoma has sometimes been arrested by early small sclerotomies around the cornea, or by iridectomy. Extirpation of the superior cervicosympathetic ganglion, and antisyphilitic treatment preceding iridectomy have been applied both with and without success. It is open to question whether there is not a constitutional anomaly (fetal rhachitis) at the bottom of the affection, and appropriate constitutional treatment should be considered. Acting on these considerations, I have, at the advice of Moro, prescribed, in several cases of rhachitis, the diet used in Pfaundler's Clinic for children with florid rhachitis and exudative diathesis of a moderate degree. The instillations mentioned above were kept up once or twice every evening before retiring.

This diet is chiefly vegetable, with restriction of milk. It consists of five meals a day: twice milk and water with gruel, once farina, once meat soup and stewed fruit (apple purée), and once finely minced vegetables—spinach, carrots, and cabbage in turn. Infants under five months should receive no milk, this being contra-indicated, according to Moro.

II. CONGENITAL ANOMALIES AND DEFORMITIES OF THE OPTIC NERVE AND RETINA

Knots of retinal vessels, which protrude into the vitreous and which usually arise from vessels of the optic disc, less often from a retinal vessel, are not remains of embryonal vitreous vessels, but congenital anomalies occurring in the course of otherwise perfectly normal retinal vessels.

There are also vascular bundles due to affections of the ocular fundus, with arborescent protrusion into the clear or opaque vitreous. Reis observed with the ophthalmoscope an abortive involution of such cellular formations.

Cilioretinal vessels are among the less rare varieties. Under certain circumstances—as, for example, in occlusion of the central artery of the retina—they have a favorable influence on the nutrition of the anterior retinal layers. Communication between retinal and chorioid vessels, and those between the venæ vorticosæ and choriovaginales in myopic, amyotropic, and hyperopic eyes, as well as the cilioretinal vessels and the so-called “perverse vascular arrangement,” have greater value for the history of development than for practical therapeutics.

Incipient choked disc may be distinguished from the congenital anomaly of the nerve head and retina, wherein both veins and arteries are extremely tortuous and twisted (*tortuositas vasorum*), by the fact that in the latter condition the veins are but slightly dilated, if at all.

Cyanosis of the retina is caused by congenital patency of the foramen ovale.

In high degrees of hypermetropia, a congenital condition is sometimes found, which is called pseudoneuritis. The connective tissue between the nerve-fibres is frequently so markedly developed that the vessels and the optic disc are of a cloudy red color and partly covered at the margins by radial striæ. This also occurs without hypermetropia and is sometimes hereditary.

The cause of so-called congenital amblyopia is still unknown. The affection is not dependent upon refractive anomalies, nor upon any demonstrable diseases of the eye. It is seemingly stationary and, according to Heine, associated with a small central scotoma in 90 per cent. of the cases. Perhaps it is caused by hemorrhage. Changes in the macula lutea have been rarely observed, although strabismus is frequently associated with congenital amblyopia.

Absence of a fovea centralis, which was observed by Seefelder in congenital aniridia, was also found by Fritsch in the albinotic eyes of a heredo. It seems, therefore, that the visual weakness of albinotic eyes depends also, to a large extent, upon imperfect development of the central area of the retina.

Medullated nerve-fibres are not very rare, and are visible with the ophthalmoscope. Their etiology is not clear. They are usually located on the anterior surface of the retina and in close vicinity to the optic disc. In rarer cases they form isolated spots in the retina, well away from the disc.

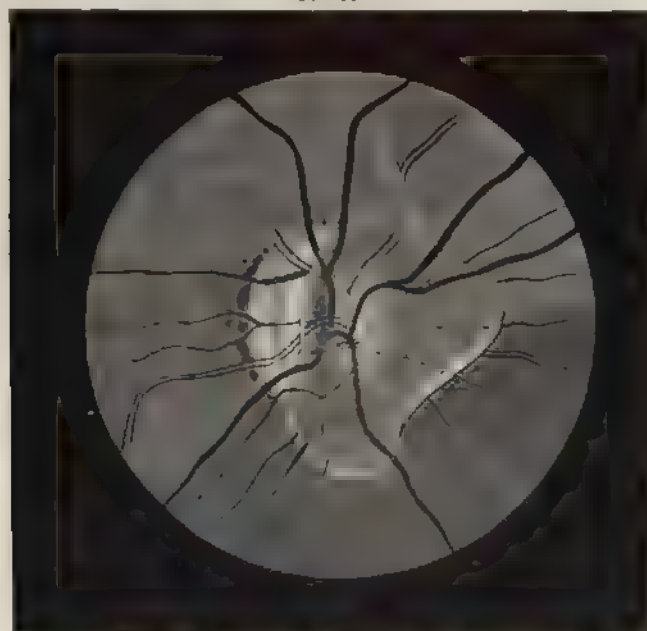
They are not congenital, according to E. v. Hippel, since the fibres of the optic nerve in the new-born do not possess a medullary sheath. In rabbits and dogs, where they are normally present, they have not appeared at the time of birth. No importance attaches to the findings, however, except that if of considerable extent they cause enlargement of the blind spot. They are said to occur in mentally impaired individuals along with other signs of degeneration.

Elschnig found in downward conus (Plate XIII, Fig. 1) a thinning and ectasia of the internal ocular membranes and sclera, in the part lying behind the posterior section of the globe, to which the conus belongs; also, a rupture between the junction of the dura and sclera and dilatation of the interspace between the sheaths in the area of the conus.

A part of the optic disc and the adjacent retina is in otherwise

normal eyes often covered by a sharply defined membrane of varying extent, which may either be impenetrable to light and of whitish appearance, or less dense and exceedingly thin, hazy, and transparent. The retinal vessels of the optic nerve in this area and the adjacent nerve seem to be covered or veiled. The contiguous parts of the optic disc and retina, however, are seen with the glasses requisite for this peculiar anomaly. In a case which I have described (Fig. 10) it was a remarkable fact that ordinary gas or petroleum illumination showed the vivid reflex of the hazy area, which was also seen by O. Becker in infantile eyes

FIG. 10.



Prepapillary connective tissue (After O. Everhusch)

which had become blind from cerebral affections. According to O. Becker, the phenomenon seems to be due to thickening of the *membrana limitans retinæ interior*.

As yet no anatomical examinations are available in regard to the changes in question, but probably they bear some relation to an unusually strong development of the connective-tissue meniscus, which was found by H. Kuhnt at the optic disc, and to a thickening of the adjacent part of the internal limiting membrane of the retina. Or perhaps these changes depend on a persistence of the posterior part of the hyaloid artery.

Aside from the clinical changes which were formerly interpreted as coloboma of the optic nerve, but what are more correctly designated as coloboma "at the optic disc" (v. Hippel), the following deformities

occur there: aplasia of the lower half of the optic nerve, and circumscribed cavities and fossæ in the head of the optic nerve, usually covered by a delicate hazy tissue. I have also observed a most peculiar form of arrested development of the optic disc, which to my knowledge has never been described. It is associated with considerable extension of medullated fibres of the optic nerve, and a high grade of congenital myopia. (Klin. Monatsblätter für Augenheilkunde, 1885.)

Aplasia of the optic nerve and retina—total absence of the nerve-fibres and ganglion-cells—has been found histologically, aside from anencephaly, in cyclopia, in microphthalmos with or without orbital cysts, and in high grades of hydrocephalus internus. Ophthalmoscopically, it has been found in the eye of a nearly full grown rabbit.

Another congenital anomaly is "color-hearing" (*audition colorée*). This may occur early in life. It consists in the mental association of colors with certain sounds. Vowels and diphthongs, names of weeks, proper names, musical sounds, etc., suggest certain colors or mixtures of colors, the deeper sounds and vowels producing dark, and the higher sounds lighter shades.

12. CONGENITAL ANOMALIES OF THE ORBIT

Dermoid cysts of the orbit (Fig. 11) are globular tumors situated in the anterior part of the orbit, and often connected with the brain by a prolonged extension. Meningoceles and encephaloceles of the orbit, which must be considered in a differential diagnosis, are usually situated in the upper nasal part of the orbit. A hernial sac, from the dura, usually can be seen to pulsate, and this, with the cerebral symptoms (vertigo, etc.), which often result from pressure, are the most important signs of differentiation. In one instance the globes were replaced by two globular structures composed of glia- and ganglion-cells, which were connected with the brain by a pedicle in all probability passing through the optic foramen. Dermoids are to be removed by extirpation. The histological composition of teratoma of the orbit corresponds to that of embryonal teratoma. This may appear at birth as a large, rapidly growing tumor. Removal of the contents has so far been successful in only one case. Usually, death occurs early.

Congenital exophthalmos is rare. It is caused by orbital hemorrhage at birth, or by a contraction of the orbit because of premature ossification of the cranial sutures. This may be associated with an increase of intracranial pressure, which pushes the globe forward in the orbit. That may even lead to dislocation of the globe.

Cohen observed a case with abnormally high vaulted skull which presented a marked exophthalmos. When the lids were lifted the entire globe could be easily grasped from above. The orbit was flat.

Enophthalmus is the result either of congenital microphthalmos or congenital insufficiency of the external ocular muscles, or of degeneration of their connective tissue and subsequent shortening.

Congenital, uni- or bilateral anophthalmos are found more rarely. Usually they are associated with other deformities, especially such as relate to the brain. A rudimentary, well-developed globe has been repeatedly found histologically in a normal orbit, although clinically it was totally absent. In unilateral anophthalmos, the other eye was either normal, or there were microphthalmos with or without a cyst, coloboma, or a high degree of asthenopia and nystagmus.

The clinical findings in typical cases are as follows: the palpebral fissure is abnormally narrow; the lids are small and can be opened only with difficulty; because of lack of support of the globe the ciliae are inverted; the orbit is very small and invested with mucous membrane; there are distinct movements in the depth of the small conjunctival sac, especially when the other eye is better developed, showing the presence of ocular muscles capable of function. At times there is purulent secretion of the conjunctiva at birth. Microphthalmos and coloboma-like anophthalmos are also accompanied by other physical deformities. The optic foramen is either very small or entirely closed.

Microphthalmos and Anophthalmos with Orbitopalpebral Cysts.—These cysts are often accompanied by other deformities. They have a direct relation to the development of the globe, and occur more often in one eye than in both. They usually appear as a large, tense tumor of the lower eyelid, less often of the upper lid, hiding the orbit to such an extent that the diminutive globe may be entirely overlooked. The following clinical facts are noteworthy: the lower lid bulges out, owing to a distinctly fluctuating, movable, globular tumor, there is slight ectropion of the conjunctiva, and the upper lid is inverted. Upon pulling the lids open, a small, firm, motile corpuscle will be seen at the end of the conjunctival sac, but only rarely distinct microphthal-

FIG. 11.



Dermoid of the orbit with exophthalmos and displacement of the bulb downward.

mos. Sometimes it can only be demonstrated by palpation, if at all, so that a clinical diagnosis of anophthalmos is often made. A clear alkaline fluid is voided upon puncture.

The frequent occurrence of coloboma, microphthalmos, a combination of both in unilateral cases, and anophthalmos of the other side, shows the close relation of these conditions. Other physical deformities may likewise occur.

The globe may be connected with the primary cyst by a scleral cyst filled with folded retina or glia. The cyst, however, is invested throughout with retinal tissue. These anomalies may have been formed in the first stage of development of the optic vesicle, or after an imperfect or atypical transformation into the optic cup has taken place; but they may also have occurred after the regular formation of the optic cup has taken place. These cases are attributed to non-closure of the fetal optic fissure, as in typical coloboma, and the globe is always better developed or nearly normal. The condition of these eyes corresponds to that of colobomatous, microphthalmic eyes without cyst formation.

Congenital serous orbital cysts, which are not connected with the globe, probably owe their origin to a transposition of the mucous membrane belonging to the nose or an accessory sinus. In Weinstein's case, an infant of eight months, the cyst had even caused an abrasion of the bone at the lower orbital margin.

The nature and cause of cyclopia are still obscure. Fortunately, most of these deformed infants die during or immediately after birth.

Deformities of the eyes, such as anencephaly, hemicephaly, and other malformations of the brain, are only of theoretical interest. Persisting hyaloid artery and pupillary membrane are of comparatively frequent occurrence.

13. CONGENITAL ANOMALIES OF PIGMENTATION

Absence of pigmentation (albinism) is always accompanied by photophobia, and often by nystagmus. The condition invades the entire eye and its adnexa, including palpebral conjunctiva, ciliæ, hair, and eyebrows. In some cases there is very slight pigmentation.

Congenital partial absence of pigmentation (circumscribed poliosis) occurs either in the ends of the ciliæ alone, or in all the ciliæ of one side. Eyebrows and hair are correspondingly pigmented.

In some cases none but the uveal pigment is absent, while the epithelial layer (external layer of the optic vessel) is pigmented, as shown by the blue color of the iris. The hair, which is either slightly pigmented or not at all, may become brown at the time of puberty, but the eyes remain unchanged.

Heterochromia, which occurs very often, is of no significance. It consists in the presence of different colors in one iris; dark areas are scattered about like islands in the lighter stroma of the iris. Or a more or less sharply demarcated sector may be much darker than the rest of the iris. In congenital heterochromia, one eye is blue and the other brown; sometimes the difference is not very noticeable, the colors being dark gray-blue against light or dark brown. This anomaly is due to the pigmentation of one eye being arrested in early life or to its later fading. The anomaly is not always without significance, because less pigmented eyes have a greater tendency to disease than normal ones, coloboma and paresis of the sympathetic having been observed. Fuchs observed in many cases a chronic cyclitis in the lighter eye. This cyclitis was characterized by the absence of injection, by strictly unilateral occurrence, delicacy of the exudate, thinning of the pupillary margin, absence of synechiæ, and frequent cataract formation. The condition seriously influences therapeutic measures and forbids a favorable prognosis in operation for cataract. Cyclitis does not set in before the age of ten years, usually toward thirty, the greatest trouble occurring in the decade between thirty and forty. It has no reference to sex, occupation, or to other diseases.

Circumscribed pathological pigment spots of the sclera, conjunctiva, iris, and chorioid are of clinical importance by becoming the possible starting point of melanotic new growths. For this reason the removal of a pigmented nævus of the conjunctiva is urgently necessary, including careful dissection of the part intimately adherent to the limbus. Pigmentation of the optic disc has been established as an anatomical fact. When seen with an ophthalmoscope it may simulate a deep pit, owing to the dark shadows cast by the margin.

14. CONGENITAL DISORDERS OF THE OCULAR NERVES AND MUSCLES

Paralysis of the motor nerves, especially of the abducens and oculomotor (Plate XX), are often caused by cerebral diseases of intra-uterine origin. Congenital insufficiency of motility is also caused by cortical injuries at birth, such as hemorrhage into the nuclear region, or basal changes, or paralysis of the abducens after forceps delivery, etc. Or there may be imperfect development or fibrous connective-tissue degeneration of the ocular muscles, or an abnormal insertion onto the globe. Imperfect nuclear development of congenital origin always affects the motility of both eyes.

Ptosis occurs relatively often. In these cases there is considerable reflex innervation of the frontalis muscle, and a pronounced backward inclination of the head, when the child attempts to open the eyes. This affection has also been observed as an hereditary disease, sometimes

in the male alone, or in both sexes; it may also alternate, the father having ptosis on the right side, and the son on the left.

In anophthalmos, microphthalmos, and encephaly the ocular muscles are usually normal, imperfect development being rather rare. Some peculiar retraction movements of the globe have been described, when the eye assumes certain positions.

Ptosis of various degrees is caused by absence or imperfect development of the oculomotor nerve, or by imperfect development and connective-tissue degeneration, or abnormal insertion of the levator palpebræ. While it usually affects one eye, it is sometimes bilateral. It is accompanied by impaired upward motility of the globe (Fig. 1), frequently less often by defects of other external ocular muscles, also by abnormal associated movements of the prolapsed upper lid, as, for example, when the mouth is opened or upon masticating, swallowing, etc. There may be a low orbital roof, epicanthus, blepharophimosis, absence of the caruncle, coloboma of the bulb, astigmatism, hyperopia, amblyopia, nystagmus, and irregular development of other parts of the body. The extent of these complications varies greatly.

Treatment is required either for cosmetic reasons, or to remedy some impairment of vision. If there is objection to operation, so-called ptosis spectacles may be worn, which will raise and support the drooping upper lid, without interfering with its action (Abelsdorf, Goldzieher, Mackness, E. Meyer, Salmonsohn). Surgical treatment consists in transmitting and enhancing the action of the frontalis muscle to that of the upper lid, or in shortening and advancing the levator, and relaxing the upper half of the orbicularis palpebræ (methods of Pagenstecker, Hess, Eversbusch, Elschmig, Wolff). Motaïs recommends the suturing of a muscle flap, cut from the middle of the rectus superior, to the palpebral cartilage, causing the globe and the upper lid to be raised together. The effect of adhesions of the levator to the superior rectus muscle must be taken into consideration, that being sometimes for evil as well as good.

No operation, however, should be undertaken too early, because congenital ptosis may undergo partial involution during the first years of life. Nor should closure of the lid be interfered with, as otherwise persistent corneal ulcers may result, such as occur in facial paralysis, thereby endangering vision.

If a 5 per cent. solution of cocaine reduces the ptosis, by contracting the tensor tarsus (Mueller's muscle) which is attached to the levator, that muscle, or a remnant of it, is evidently present.

Jerky, intermittent, or rhythmical and periodical twitching of the upper lid has been observed several times in congenital or early paralysis of the levator, which was accompanied by disturbances in the innervation of the external ocular muscles.

Congenital ptosis should not be confused with a markedly developed, drooping fold, so-called adipose or atonic ptosis. Blepharochalasis (Fuchs) or skin atrophy, which is confined to the upper lids, results from repeated inflammatory swelling and loosening of the tarso-orbital fold. This usually commences at puberty, but may so increase in the course of years that in order to improve vision it may be necessary to excise part of the abnormally flabby skin which droops like a sac over the margin of the upper lid. This is followed by suturing to the tarsal margin. Schmidt-Rimpler observed an adipose hernia in the orbicularis of the upper lid of a girl of 19, which was probably caused by a slight congenital defect arising from the orbital septum. As a result the upper lids looked like œdematous ridges, the upper fold drooping more than usual.

Hummelsheim advises transplantation of muscle in congenital defects of motility of the ocular muscles, as, for example, parts of the superior and inferior recti, in the absence or imperfect development of the external rectus. In a congenital paralysis of the abducens on the left side I performed a very successful operation by advancing the tendon of the external rectus as closely as possible to the temporal corneal margin by means of three sutures. One suture was placed in the horizontal meridian, the second and third subconjunctively toward the lower tendinous margin of the superior rectus and the upper tendinous margin of the inferior rectus, respectively. The tendon of the internal rectus was also divided. In order to preserve as far as possible associated vision of both eyes to the right, I carefully folded the bulbar conjunctiva by three very superficial sutures. A figure-of-eight bandage, covering both eyes, was applied for seven days, followed by careful removal of the threads, some of which had already loosened.

Uni- or bilateral facial paralysis as an hereditary affection is rarer. It may appear either alone, or in conjunction with paralysis of the nerves of the ocular muscles or their cerebral nerves, trigeminus, etc., or with other congenital anomalies of the eye, ear, or other parts of the body.

Absence of Bell's Phenomenon upon Closure of the Lid.—A congenital paralysis of the right oculomotor nerve, with a corresponding change in the size of the right pupil, was observed by v. Michel in a six-year-old girl. The facial paralysis was either congenital or developed at a very early age. Contraction of the orbicularis caused narrowing of the pupil with simultaneous slight spasm of accommodation.

A right, congenital, widely gaping palpebral fissure, accompanied by slight exophthalmos and hyperæmia of the right side of the face, was observed by v. Michel in a neuropathic nine-year-old boy. There were no associated movements of the upper lid when looking downward. The superior rectus was partially paralyzed.

The constant movement of the globe, described as nystagmus (horizontal, vertical, and rotatory), is particularly intense in albinism, where it is frequently accompanied by congenital or early acquired blindness. Consanguineous marriages are mentioned as one of the causes.

15. OCULAR ANOMALIES OR AFFECTIONS DUE TO ANCESTRY, HEREDITY, OR CONSANGUINITY

Under this heading may be mentioned high degrees of hyperopia, and myopia or predisposition to myopia, strabismus, nystagmus, albinism, macular disease, optic neuritis or tendency thereto (Leber), and retinitis pigmentosa. The latter disease begins with night-blindness (hemeralopia). Later on there is concentric contraction of the visual field, due to annular scotoma. Complete blindness ensues, the result of optic atrophy.

A certain family by the name of Nougaret furnishes an interesting record of inherited stationary night-blindness. It existed with normal visual acuity and normal visual fields, without fundus changes which would be recognized with the ophthalmoscope, and showed no exacerbations as time went on. There were two thousand one hundred and sixteen persons in this family. Among two hundred and fifty-five persons who belonged to the affected branches of the family, no less than one hundred and thirty-five suffered from night-blindness. This category also includes inherited word-blindness and amaurotic idiocy. This latter condition has been observed to accompany a rapid atrophy of the optic nerve without paralysis, either at an early age, or between the sixth and seventh years. It may present the picture of retinitis pigmentosa with distinct constriction of the arteries (Stock), but without noteworthy affections of the nerve. There is also that form of hereditary atrophy of the optic nerve which attacks the male sex preëminently, and is characterized by an affection of the papillo-macular bundle, seriously interfering with central visual acuity. Peters had attributed it to a congenital defect in the germinal layer. Ocular affections in which consanguinity is particularly concerned include inherited color-blindness, either partial or total.

In total color-blindness there is only quantitative differentiation of colors, everything appearing gray in gray. This is exceedingly rare. It is usually associated with considerable reduction of central visual acuity, photophobia, and nystagmus, or with a distinct central scotoma. These patients perceive after-pictures of their impressions in the region of the fovea, similar to the same perceptions in persons with normal vision, except that the excitation of the fovea occurs more slowly than that of the adjacent parts of the retina.

Partial color-blindness is inherited relatively often. Its variations are blindness for red, green, and violet (Helmholtz); red and green, with relative vision for blue and shortened spectrum; red and green, with relative vision for yellow and shortened spectrum; yellow and blue

(Hering); red; green; and violet (v. Kries). It is well known that the normal daughters of a color-blind father procreate color-blind children, and the affection has been thus observed in succeeding generations. The male sex is more frequently affected than the female. No observations have as yet been made in regard to the heredity of the rare yellow-blue blindness (tritanopia). Color weakness (abnormal trichomasia), which occurs under similar hereditary conditions, is also of practical importance, small colored objects being discernible only with great difficulty. Recognition of red and green, for instance, occupies a long time. The contrast of colors is increased, so that colored lights standing behind each other are named correctly, but those standing side by side only with hesitation or incorrectly. Practical tests of the color-sense are those with Daal's or Helmgren's wools, W. v. Nagel's color plates, and Pflueger's method of color contrast. The examination is not always easy, since color-blind children with an excellent sense for form have learned by experience to recognize and name correctly even very subtle shades. Simulated color-blindness is easily detected by Stilling's pseudo-isochromatic tables.

It sometimes happens that in pronounced, bilateral hereditary ocular affections only one eye will be affected,—as, for example, in myopia,—while unilateral retinitis pigmentosa or Daltonism is rare.

Inherited anomalies of the eye in juvenile dementia are likewise of importance. Gelpke found normal vision in only 27.5 per cent. of five hundred and seventy-eight weak-minded, demented, and idiotic children. The affections observed comprised amblyopia without great anomalies of refraction, astigmatism, hyperopia, strabismus, deformities, and acute or chronic inflammations. Among idiots, there are no less than 32.8 per cent. with inherited defective vision.

Pilcz and Wintersteiner observed a great many cases of high degrees of myopia in patients suffering from psychoses, where hereditary taint plays an important part, and also other hereditary ocular affections in paranoia, in periodical psychosis, and feeble-mindedness.

By means of individual and family investigations extending over various generations attempts have been made in recent times to establish the exact way in which ocular defects are inherited. Especial efforts have been made to determine whether blood relationship is of any particular significance, whether these anomalies strictly conform to Mendel's law, and how the immunity of any intervening generation can be explained.

The following important points have been established as a basis for future hygienic procreation: The rules of "direct" heredity of dominant symptoms are followed in cases of stationary congenital night-blindness, distichiasis, ptosis, nystagmus, coloboma, glaucoma, various forms of congenital cataract, nodular and cancellatous keratitis, and convergent strabismus associated with hypermetropia and amblyopia.

Nettleship and Lawford found that glaucoma occurs earlier in the younger generations than in the older, and that in the same generation the younger members of a family are affected earlier than are the older members.

Among the conditions which are distinctly hereditary are especially those which relate to anomalies of refraction, and to imperfect development of the optic nerve and retina. The children of ametropic parents inherit these anomalies twice as often as do the children of parents who have normal eyes. A. Lutz reports a family in which the anomalies were restricted to the children who had brown eyes. That is, the brown color was a dominant hereditary characteristic. In these the ectodermal and mesodermal pigment of the iris is developed, as against the blue or gray eyes. According to A. Steiger, the tendency to corneal astigmatism in children decreases somewhat in proportion to the age of the parents.

Inherited defects of the cornea or of the lens, in other words, anomalies of refraction with certain forms of color-blindness, are often accompanied by various anomalies of the nervous system. Typical examples of this are seen in nystagmus, photophobia, and allied conditions.

Again, inherited defects and deformities of the eye, such as albinism, color-blindness, astigmatism, medium and high degrees of myopia, lenticular opacities, medullated nerve-fibres, and slight irregularities of the optic nerve, such as downward conus, may occur and be inherited in conjunction with imperfect cerebral development, mental inferiority, epilepsy, and endogenous psychosis; or they may find expression in amaurotic family idiocy or mongolian idiocy, with oblique position of the palpebral fissures.

In retinitis pigmentosa consanguinity is without doubt an important factor. This is also true of albinism, of congenital cataract and high degrees of corneal astigmatism. Such instances of heredity have been cited by Hutchinson, Nettleship, and Doyne. A. Lutz observed it in four girls belonging to one family, the condition resembling a distinctly pronounced central macular affection. Total or partial color-blindness and optic atrophy are also hereditary, collaterally or indirectly with the omission of a generation.

According to Martius, inherited red-green blindness is on the increase. He regards Daltonism as a negative variant, dependent upon the absence of certain physiological determinants in the chromosomes of the fertilized ovum. He looks upon myopia as belonging to the category of branch-deviating properties.

No importance seems to have been attached to unilateral color-blindness in the genealogical researches (O. Becker).

Congenital blindness is principally due to inherited syphilitic affections of the optic nerve, retina, and chorioid, to the early forms of pig-

ment degeneration of the retina, to congenital hydrophthalmos and to cataractous degeneration of the lens in consanguineous marriages. It is attributed less often to deformities of the eye in the actual sense.

According to Heine, "congenital amblyopia," which is not sufficiently explained by the ophthalmoscopic picture nor by the refractive media, is caused in 90 per cent. of the cases by a central scotoma. This may be difficult to find, and may change in intensity and extent. But it generally prevents, and in such a proportion to the decrease in the visual acuity as to account for the degree of congenital amblyopia. According to W. Lohmann, the ultimate cause is that in these cases the perceptive elements are farther apart than normally. Where this exists he has found a difficulty of fusion such as exists in persons with normal vision in the peripheral parts of the retina. The affection is nearly always unilateral. High degrees of bilateral amblyopia are very rare.

The question of origin, however, is still an open one, especially its relation to a possible injury at birth; because it is very improbable that extravasations of blood occurring during labor would always be absorbed without leaving a trace.

Congenitally blind individuals, who have been successfully operated upon, learn to understand their new impressions with varying degrees of rapidity. Those who become blind at an early age will learn quicker in proportion to the extent to which they recall their first optical concepts.

The causes of blindness acquired in infancy, which still amounts to 25.6 per cent. of the total number of the blind, vary according to country and period. In Siberia, for instance, the first place, after trachoma, is occupied by smallpox, which in Germany stands last. The principal causes of blindness in the first years of life are: ophthalmia neonatorum; "scrofulous" corneal ulcers which have run an unfavorable course, as after measles; ocular diphtheria; severe trauma of one or both eyes (especially the dangerous lime burns); and sympathetic involvement of one eye after injury to the other. Such an affection is apt to run a particularly severe course in infancy.

It is only during the last decades that the relation of early blindness to weak-mindedness, and ocular defects—such as high degrees of myopia or weak vision, etc.—to mental inferiority has been investigated. Sometimes blindness occurs simultaneously with congenital weak-mindedness and sometimes the case is one of the mental backwardness of generally immature children, who have acquired blindness at an early period. Attempts have been made to improve, exercise, and utilize any possible visual remnant by the use of appropriate glasses, and the visual remnants in these children should be tested at as early a time as possible, precisely as hearing remnants are tested in children who have become deaf at an early age. Considering the absence of the simplest conceptions in the

child's mind, this is a difficult if not almost impossible task. Similar conditions prevail in testing for color perception, which demands a good visual memory, and is therefore at the same time valuable for determining the intelligence of weak-minded children. The visual acuity of feeble-minded children was found in general to be less than that of normal children, and the number of weak-minded children with red-green blindness was found to be greater than of those mentally normal.

The percentage of myopia in all groups was unexpectedly small. In children with impaired intellect the ocular defects consist chiefly in anomalies of refraction.

According to Gelpke, one hundred and eighty-nine cases of impaired vision out of two hundred and ninety-four could be considerably improved by proper spectacles. Compensation of these defects by correction, and systematic attempts to exercise the amblyopic eye with the better eye closed, are important from an educational point of view, because by improvement of the visual power children are rendered more receptive to sensory impressions of all kinds.

I have repeatedly noted that after enucleation of one eye—because of intra-ocular tumor, serious trauma, etc.—the vision of the other eye was considerably increased by careful correction of the faulty refraction after the patient had become accustomed to wearing spectacles. For example, in a young man of twenty-five, with corneal astigmatism, his central vision, which had been reduced to one-fourth, rose to 6/6. If, then, impaired vision can be improved in an eye which has passed the stage of development, this must be applicable with greater force to younger individuals. However, the expected increase may fail to occur.

Owing to their congenital defect children born blind learn to speak much later and more slowly than normal ones. Development of speech is, of course, slower still in those cases—fortunately rare—which are born deaf and blind. The outlook is more hopeful when children become deaf and blind at a later period, even if before speech has been completely acquired. Girls are relatively oftener so affected than boys. There are several cases on record of complete deafness and blindness, deafness with remnants of sight, or blindness with remnants of hearing power.

As to etiology, Bruehl found hereditary syphilis in one-half and meningitis in one-fourth of the cases; 60 per cent. of the deaf and blind were females. According to Bruehl, one-third may be saved by timely diagnosis and early specific treatment. According to the same authority, instruction is best imparted in special institutes for children who are too deaf to follow the lessons in institutions for the blind, and too weak-sighted for instruction in an institute for deaf-mutes.

Such institutes are the special school at Venersborg, in Sweden, and the "Deutsche Taubblindenheim" in Nowawes, near Berlin.

II. DEVELOPMENT OF THE INFANTILE EYE

THE eyelids of the infant present no peculiarities.

The position of the infantile orbit is horizontal. That is, an imaginary axis drawn through the middle of the facial aperture of the optic canal lies almost completely in the horizontal plane. The divergence of the orbits, which increases with age, is of importance in relation to the position of the globe.

The orbit is relatively larger in the female than in the male, while asymmetrical development is about equal in both sexes. According to Merkel and Kallius, considerable differences develop with increasing age. A frontal section behind the orbital margin in the adult approaches the shape of a circle, while the same section in the cranium of the new-born assumes the shape of a longitudinal oval. As growth proceeds, the median upper parts of the orbit alone remain substantially unchanged in their proportions. The growth of the orbit participates in that of the entire face, which is at first principally downward; as childhood advances and the face becomes wider, the orbital development, too, is chiefly lateral. The height of the facial aperture of the orbit increases so rapidly that in five-year-old children it is only 2 mm. short of complete development; at seven years of age this limit is usually attained, but the shape is roundish, owing to still undeveloped width.

The canal of the optic nerve is wider and more irregular in children than in adults. The fissures in childhood are broad and roomy, owing to the narrowness of the temporal part of the sphenoid bone.

The changes of the orbital shape following shrinking of the anterior section of the globe or after atrophy or loss of the eyeballs, owing to phthisis, enucleation, or exenteration, are rather important and in the main concern the anterior segment of the cavity. The soft parts in the posterior section remain more or less well preserved. The longitudinal diameter remains unchanged, the roof shows a slightly irregular flattening of the concavity, and the floor a slight raising. The facial orifice of the orbit, however, undergoes the greatest changes, its height being relatively diminished, while its width remains the same.

These changes may be distinctly seen in early childhood, even within a period of ten weeks.

According to Merkel and Orr, the shape of the eye in the new-born is much distorted. The nasal half somewhat resembles the adult shape, but the posterior part of the lateral half of the globe has a considerably greater curvature. Accordingly, the visual axis, or the line between the apex of the cornea and the fovea centralis, has a totally different posi-

tion from that in the adult. This is for the reason that in the infant the distance of the fovea centralis from the entrance of the optic nerve is about the same as that which exists in the adult.

The anterior part of the sclera is relatively as thick as in the adult, while it is of relatively double thickness in the vicinity of the optic disc. The part of the sclera which is visible between the lids is so thin during childhood that the pigment of the chorioid shines through with a sort of bluish transparency in delicate children.

In infants the cornea is relatively larger than in the adult. Some difference in its thickness also is not improbable. The anatomical findings in this respect do not admit of conclusive proof, as they may be illusory, owing to an indifferently preserved condition of the eye.

The layer of the eye in the new-born is characterized by an absence of pigmentation, except that the vicinity of the optic nerve shows some pigment deposits. Owing to this absence of pigmentation of the stroma, the color of the iris in the infant presents all varieties of shade from light blue to blue-gray, although it has been stated that here and there children are born with brown irises. They soon begin to darken, and within a few weeks a rather dark brown color may appear. In other cases it may require from several months to two years before the iris has attained its definite color and development. Even in adults a slight increase in pigmentation is quite an ordinary occurrence. The processes of the stroma cells at the time of birth have been found microscopically to be much more slender than in adults. The other parts of the medial tunic probably follow the same course in pigmentation as the iris.

The anterior surface of the iris, which in the new-born is pressed forward by the lens, presents the same material condition in childhood as in later years, although less distinct. The stroma is at first very thin. The posterior surface of the iris contains crest-like prolongations of the ciliary processes, which traverse the iris radially and later disappear. In children the pigment membrane on the posterior surface of the iris is usually so strong that it may be completely detached by maceration.

Congenital variations of the surface of the iris occur often in the form of small nodules which are of a light color and project somewhat above the surface of that membrane. These are far more frequent in the lower than in the upper half of the iris. Sometimes these nodules appear to be loosely adherent to the iris near the ciliary margin, rather than at the pupillary margin. One can sometimes count ten or twenty, or even more, though they are then not uniformly scattered over any particular area.

The pupil is not often in the centre of the iris, but lies inward and downward by about a sixth part of its diameter.

The sphincter pupillæ in the infant has the same width as in the adult, but has not yet attained its thickness. On the contrary, the

YASSEL: 3MA:

ciliary body is half as thick and not so long as in the adult. The individual meridional and circular fibres of the ciliary muscle, although varying in bulk, can already be recognized. The lens is disproportionately large during fetal life, its development anticipating that of the entire globe. It occupies almost the entire bulb, leaving but a small space for the vitreous behind it. It is globular in shape. In the new-born, too, it is still very large in proportion to the space occupied by the globe, and, according to E. v. Hoppel, also rounder in shape than it is later. The lenticular margin in the infant is almost as sharply delineated as in the adult. Similarly, the posterior surface has a greater curvature than the anterior. The sagittal diameter is scarcely smaller than in the adult, while the equatorial circumference is still considerably less. Therefore, the growth of the lens, which continues until over thirty years of age, is restricted to the enlargement of the circumference. In childhood it consists of a soft but inelastic substance; a real nucleus is not yet demonstrable, the centre being almost as soft and osmotic as the periphery. The elastic, lenticular capsule grows thicker in the course of years.

The zonula is firmly adherent in the new-born to the vitreous, except at its anterior part. Its fibres extend from the ora serrata to the posterior surface of the iris. Their number, however, decreases considerably in time. The extensions of the ciliary processes to the iris disappear, together with the foremost zonula fibres, in the course of childhood. As age increases, the number of zonula fibres, which are attached to the lens in a dainty denticulation, gradually decreases and the single fibres become thicker.

The vitreous changes, during the first year, to a more compact cortex, and a nucleus is formed by a fold, consisting of easily torn and even deliquescent tissue, which in very advanced age seems to disappear, save for a few remnants.

The ora serrata has the same macroscopic appearance as in the adult, except that the visible serration of the retina is somewhat less pronounced. A physiological excavation of the optic disc is present even in the new-born.

On the other hand, the distinct thickening of the margin of the fovea centralis formed in the adult is absent in the new-born, so that the glistening reflex seen with the ophthalmoscope of later life is not present in the infant's eye. The fovea in the new-born (Konigstein), though still functionally defective, undergoes considerable changes during the first weeks of life. This is especially by the formation of the so-called outer fibrous layer, by increase and development of the cone bodies at the floor of the fovea, and by involution of the inner layer.

The optic nerve is quite fully developed, its width being only a few millimetres less than in the adult.

Its post-embryonal growth consists principally in the addition of medulla in the fibres, as the medullary sheaths have only begun to appear at birth. This explains why the striking change of the color at entrance of the optic nerve of the adult is absent in the new-born. It is due to the marked thinning of the nerve at that place. The diameter of the central vessels increases considerably later on (Hertel). The vessel walls are about half as thick as in middle life.

The weight, shape, and size of infants' eyes may vary greatly. This is true of the emmetropic eye of the adult also, as one may find large eyes with weaker vision, and small eyes with a strong optical system, the parallel rays in each case falling upon the retina as a true focal picture.

While the weight of the entire body increases twenty-one times during the period of growth, and that of the brain 3.76 times (Vierordt), the weight of the eye until fully grown increases 3.252 times, and its volume 3.292 times (L. Weiss). The greatest and most rapid growth in both directions occurs in the first seven years, after which it is both absolutely and relatively much slower than the growth of the body. The diameter and circumference of the eye, according to L. Weiss, are as follows: The proportion of the vertical, horizontal, and sagittal diameters is 1.0 : 1.04 : 1.065. The vertical and horizontal diameters grow in about the same ratio; the increase of the sagittal diameter is somewhat less, so that the proportion between the new-born and the adult is as follows: Vertical diameter 7 : 10.76, horizontal 7 : 10.69, sagittal 7 : 10.19.

In this way the juvenile eye grows more globular up to the ninth year. This difference increases as the child grows older, bringing the eye to its final shape, in which the horizontal diameter is equal to, or larger than, the sagittal, while the vertical diameter remains somewhat smaller than the other two. This is in spite of its greater growth, since at birth it was considerably smaller than the sagittal.

The vertical circumference of the eye in the new-born is proportioned to that in the adult as 1 : 1.4888, the horizontal circumference as 1 : 1.454, and the equatorial circumference as 1 : 1.504.

The equatorial circumference of the eye was calculated by L. Weiss as 49.3 in the new-born and 75.55 in the adult.

Up to the second quarter of the third year the growth of the optic axis very nearly corresponds to that of the brain. Weiss and Halben have established the following measurements: The length of the optic axis in the new-born is 16.6 mm.; it grows in the first month to 17, in the second to 17.5, in the third to about 18, in 6 months to 18.6, in 12 months to 19.5, in 18 months to 20.3, and in 3 years to 21 mm. From this period onward the growth of the axial length is considerably retarded.

At the age of fourteen to fifteen years it amounts to 22.3 mm., while in the adult, according to Weiss, it measures about 23.85 mm.

The ocular region of the normal infant presents nothing of particular interest, save for slight superficial injuries of the skin, which may be due to forceps delivery. There is some slight œdema of the lids and very slight hyperæmia or icterus of the conjunctiva, which may also be infiltrated with slight ecchymoses. There is often a light, diffuse, grayish opacity of the cornea, which is rarely pronounced, and disappears more or less rapidly in the course of a few days. Generally the anterior chamber is rather flat. Residues of the pupillary membrane are relatively often present, either in one or both eyes. Some of the threads traversing the pupil are so delicate that they can be recognized only with oblique illumination.

Both the lens and the vitreous are quite transparent. The ocular fundus is always lighter and less pigmented than in the adult, and resembles that of light blondes; the chorioidal vessels are often distinctly recognizable and the intervacular spaces are very light. The region of the macula lutea is more uniform and of a rather dark tint, but rarely much pigmented. The retinal reflexes, which can sometimes be seen in older children, are absent, nor is the reflex stria of the retinal arteries as distinct as in adults.

The optic disc has either a distinct blue-gray or pale red coloration, according to the illumination used; its shape is almost circular, the vertical axis being only slightly longer than the transverse. If the transverse diameter is considerably reduced, a narrow pigment fringe can be seen at the outer margin of the disc, usually with a narrow scleral attachment. The optic disc is often sharply outlined by a more or less substantial pigment line. Otherwise the ocular fundus presents a number of individual physiological peculiarities, which do not deviate in the main from the ophthalmoscopic picture in the adult.

Hemorrhages of the retinal veins in one or both eyes, usually due to diapedesis, less often to rupture, are of exceedingly frequent occurrence. They are usually located in the region of the posterior pole, less often on the optic disc and in the parts of the retinal periphery visible with the ophthalmoscope. According to v. Sicherer, the right eye alone is affected, or to a greater extent, by the first occipital presentation, while the left eye more especially in the second occipital presentation. Two exceptions to this rule, however, have been observed when the caput succedaneum was on the one side corresponding to the ocular hemorrhage. Retinal hemorrhages of childbirth appear either as roundish, irregular, dark red spots of varying size, or in a distinctly radial or straight arrangement. Sometimes they are isolated, in other cases they are so numerous and diffuse, extending to the periphery, that but few areas of the ocular

fundus are free from them. They are speedily absorbed without leaving any of the white spots, such as are often seen after other retinal hemorrhages. These hemorrhages are attributable to marked venous stasis during birth. If, however, there is a hemorrhage of the chorioid in the macular region, any coexisting retinal hemorrhages must receive serious consideration, especially if there is an hereditary predisposition to hyperopia of high degree. Then hemorrhages may give rise to congenital amblyopia without actual ophthalmoscopic findings later. If the hemorrhage is chorioidal, congenitally atrophic foci in the pigment epithelium may follow as well as those atrophic changes in the centre of the ocular fundus which together pass later for a central chorioid coloboma (Plate XIX). Such a causal connection is still more probable, if simultaneously with retinal hemorrhages there is hemorrhage into the anterior chamber and vitreous, such as has been observed when the eyes were really injured by forceps delivery but without much apparent contusion of the globe.

It is not likely, then, that an abnormally pale color of the optic disc, coupled with unusual thinness of the retinal vessels and intense infiltration and opacity of the papilla and its adjacent area, will disappear without leaving any trace. As a matter of fact, in two cases *v. Hippel* found anatomically very extensive hemorrhages in the suprachorioidal space, as well as in the disc and retina. This is also supported by the central scotomata found by *Heine* in the majority of 100 cases of congenital amblyopia.

An ophthalmoscopic picture which I have often observed is characterized by peculiarly formed extensive pigmentation and connective-tissue development in the region of the optic disc and the surrounding retina. It is accompanied by more or less pronounced changes in the pseudomembrane of the retinal vessels, especially increased tortuosity and connective-tissue sheathing. This picture is likewise due to hemorrhages of the optic nerve and its sheaths, with destruction of the optic disc. In most cases this is caused either by compression of the orbit, in the passage of the foetus through a constricted pelvis, or by application of the forceps. These vascular changes at once preclude any confusion with medullated nerve-fibres. In the differential diagnosis we should also consider retinitis proliferans, and the results of tension in the optic nerve.

Certain cases of strabismus and insufficient fusion will also have to be referred to congenital defects. *Panas* attributes congenital concomitant strabismus to meningeal interpartum hemorrhages comprising the muscle nerves of the eye in their intracranial course. As a matter of fact, *Scrinii* and *Pinard* have often observed in the new-born, especially in primiparae cases, alternating periodical strabismus, which usually disappeared later, but which persisted in a few cases.

According to Schneller, the volume, thickness, width, and length of the recti externus and internus are considerably smaller in the new-born than in the adult. According to L. Weiss, the insertions of the four recti muscles are 7 mm. nearer the optic disc, and a comparison of their relative distances with those in the adult shows the following figures: 1 : 1.5 : 1.4 : 1.36, as against 1 : 1.36, 1.4 : 1.6 respectively.

In regard to the behavior of the muscle insertions in the growing and fully developed eye, the following facts are of importance: The insertions of the recti externus and inferior, which are the narrowest at the time of birth, increase more, relatively, than the two largest, the recti internus and superior. Sometimes the line of insertion is nearly straight, sometimes more or less curved, sometimes quite irregular. In exceptional cases there is a separate second insertion, which is more or less developed and lies behind the regular insertion. Its presence must be suspected if, in an operation for strabismus, squinting continues, in spite of a complete division of the affected muscle.

The distance from the insertion to the corneal margin is largest in the superior rectus, smallest in the internal rectus; the external and inferior recti are equidistant therefrom, although occasionally one is a little nearer than the other. Oblique insertion of the internal and external recti also often occurs, but the deviation is usually slight, and compensated for by the fact that they diverge in opposite directions. It also happens that the lines of insertion of these two muscles do not run symmetrically to the horizontal meridian and so, instead of striking the centre of the meridian, they are changed in such a way that either the part above the horizontal meridian is smaller than that below or the tendon of the internal rectus is displaced downward and that of the external rectus upward. There may be marked variations in either eye of the same individual, sometimes amounting to diametrically opposite conditions. Similarly, the insertions of the superior and inferior recti are often asymmetrical in the new-born as well as in the adult. The direction of the lines of insertion of the oblique muscles has been found to be subject to considerable variation. The distance of the internal rectus from the corneal margin increases with growth more than that of the three other straight muscles. On the other hand, the posterior section of the eye, beginning at the insertion of the rectus muscles, develops regularly in emmetropic eyes.

Variations of the ocular muscles are very rare.

The causes of the spontaneous cure of convergent strabismus are not yet settled. It may be due to a change of the orbit in relation to the growth of the skull and face, or to an increased divergence of the orbit, as L. Weiss assumes. The anterior half of the bulb may grow more than the posterior, or there may be an unusually strong development

... upon the growing eyes of ... are slightly, though per- ... being 1/15 to 1/30 part of

... in newly-born rabbits caused ... up to one-third of the volume. ... and eighteen non-operated ... showed that the non-operated cases ... corresponding to the respec- ... there were reductions amounting to ... year, and to 0.32 mm. from the

... operation was even more striking when ... or when there was a prolonged inter- ... on the first and second eye.

... established whether early iridectomy of the ... by a demonstrably smaller size of the ... iridectomy on the right eye of two boys, ... months because of adherent leucomata, and ... these patients at the age of seven years ... retardation in growth, as compared with ... with Hertel's exophthalmometer gave ... measurement of the radius of corneal cur- ... of Javal-Schiötz (which was exceedingly ... children's restlessness) showed that in one of the ... reduction of 0.25 and 0.05 mm. This should be ... of the leucomata and the corneal astigmatism. In ... the physiological width had increased by 0.095 mm.

... the removal of juvenile cataract, the considerable ... properties of the growing lens, which had long been known ... have been confirmed by Wessely's investigations, are worthy ... However, after an experimental discission and sub- ... a regenerated, almost clear lens was considerably ... of the control eye.

... not probable that the post-operative reduction of the size of ... should be attributed to the consequent increase of tension of the ... and a centripetal pulling on the ciliary processes?

... observed by C. Schweigger, and described by me in the ... *Wochenschrift* for 1901, proves that such traction on ... together with elongation of the ciliary processes, does ... in the human eye. Should this prove to be a regular occurrence, ... would follow that in operating for congenital or infantile cataract ... the anterior capsule should be liberally opened in evacuating the lens,

that absorption of the contents should be promoted by the application of heat, etc., and that a liberal division of the posterior capsule should be made if necessary to retard regeneration of the lens with its sequelæ. Care should, of course, be taken not to injure the vitreous; and any pulling of the ciliary body should be avoided. Therefore it is advisable to perform the operation under anæsthesia.

The act of seeing being a psycho-physical process, it depends upon good development of the eye and brain. An infant at the time of birth, however, possesses only a rudimentary part of the cerebellum, connected with the spinal cord by a number of ganglia near the base of the skull. All animals possess this rudimentary brain. The anatomical function of the cerebellum is not yet fully developed in infants; it is not particularly undersized, but the delicate development of the numerous tracts is still missing. If, therefore, the eyes of the new-born follow the light, it is principally a reflex movement which in a very short time is aided by turning of the head.

Thus, the new-born infant perceives impressions with the eye, the optic nerve, and the rudimentary brain. He will, however, fail to understand what he sees until the optic impressions are often repeated, and all of the complicated cortex and nerve tracts which are necessary for the mental understanding of the optic impressions have been developed. Consequently, our ordinary movements of the lids and winking of the eyes, caused by retinal fatigue, are reflex manifestations, which in infants are not yet frequent and pronounced.

Vision, and the development of the correlated cerebral tracts, which begins at the time of birth, will be missing, in the absence of optic impressions, as in congenital total atrophy of the optic nerve. The psycho-physical functions will develop more slowly if the optic impressions are impaired, as, for instance, in congenital cataract.

The behavior of the pupils in the infant and during the first year of life is instructive. Thus, Pfister found that sensory dilatation of the pupils is never present in the first four weeks. The maximum degree possible is not reached until the sixth year. Bartels states that strong illumination of the pupils of a waking infant causes but very slight reaction, the contraction amounting to no more than 3.5–2.5 mm. It requires very intense illumination to cause a contraction to 1.5 mm., such as occurs in adults over fifty years of age. With very weak illumination the pupillary diameter never exceeded 5 mm., the average being 4 mm., as against 5.5–8 mm. in middle-aged individuals. Bartels found that the pupils contracted to 1.5 mm. during sleep, while H. Gudden usually found them much less contracted than in adults (2.2–2.5 mm.). Both Bartels and Gudden have observed that the pupils dilate very slowly upon awakening, and they never exceed an average width of

3-3.5 mm. The dilatation, however, is more rapid and a little more extensive when the infant is shaken or patted upon the back.

From about the third month the pupils are somewhat more contracted during sleep, and between the sixth and seventh months miosis below 1.5 mm. has been observed. The lightning-like, extensive dilatation of the pupil in adults upon awakening is only gradually developed, and at the age of one year is yet far from complete.

The first pupillary reflex is reaction to light, which, according to Pfister, is followed, between the second and the fourth months, by reflex winking, and later by reaction to skin irritation. Reaction to mydriatics, even in conjunction with cocaine, is slighter during the first and second years than in adults, because the factors causing the dilatation are not yet powerful. The pupils are not contracted during sleep in the first weeks of life, because, according to H. Gudden, of imperfect development of the medullary sheaths of the oculomotor and optic nerves. For the same reason the connection between the centres of the optic nerve and that of the sphincter of the iris is as yet defective in function. Miosis during sleep is not distinctly present before the third or fourth month. This means that when, with the exception of the pyramids, the medullary development in the area of the crus cerebri is nearly or entirely complete, the medullary sheaths and the ganglion-cells are still only in the stage of development over large areas of the cortex. For this reason miosis may be attributed to an irritation or overactivity of the lower tracts of the oculomotor, at least in the area of its nucleus in the corpora quadrigemina. This is also indicated by the fact that the eyes of new-born children roll distinctly upward in sleep.

The sudden maximum dilatation of the pupils is a cortical reflex of the cerebrum. It does not occur until conscious movements of the mouth, arms, and hands are made and the first complicated associations of ideas are practised.

E. Hering has investigated the question whether the physiological act of vision and the perception of space is congenital or acquired. Apparently each act of distinct vision is a component part of the perception of space. Thus infants with congenital or early acquired dense opacities of the media can only distinguish light and dark. They cannot state from which side the eyes are being illuminated, although, like individuals with normal eyes, they are able to localize the so-called pressure phosphene, usually consisting of a light disc with a dark centre.

Associated ocular movements can be observed in the new-born in the waking-state, and an eye with congenital or early acquired blindness carries out the same movements as the normal eye. In the first days of life, unilateral or dissociated movements are sometimes observed. This points to subcortical innervation of the ocular muscles, aside from

the usually dominating cortical centres; the movements resulting therefrom need, of course, not be associated.

The "physiological incongruity" of the retinae, slight though it may be, points to a congenital difference in the impressions of space and direction obtained from various parts of the retina.

The binocular perception of depth is likewise a function of vision depending upon congenital conditions. The two eyes of a new-born infant do not form two pictures any more than do those of an adult. All simple and space vision is congenital, as otherwise an infant would have to search for the two pictures belonging to each other, fuse them and arrange them according to the dimensions of their depth. The same is true of the oculomotor apparatus, the associative use of which needs not be specially acquired. At the same time, habit and use in all organs, particularly the eyes, have a powerful influence upon their function. Although the structure of the eye and its central connections must be assumed to be as necessarily congenital as is the sensation of space, the latter does not display any essential associative function until it is developed from the relation of the objects to our mental organs. The surrounding world does not cross the threshold of the awakening infantile consciousness until the child has learned to realize all mental impressions as belonging or not belonging to his own body. Thus, the correct position of the eyes, when looking at an object, is not acquired until the first year, and the globes are often turned inward, even sometimes as if the object looked at were on the tip of the nose.

The fact that the new-born gradually learn to distinguish light-sensations, demarcated light-areas, to recognize shape, distance, and colors, has been observed in infants who suffered temporarily from amaurosis due to blepharospasm, or who recovered their sight through an operation after congenital or early acquired blindness. In two cases of Uhthoff's there was a remarkable reduction of nystagmus; in another case a hitherto apathetic boy acquired great vivacity.

Normal-sighted individuals, when looking straight ahead, will simultaneously perceive all objects with both eyes, except those located at the extreme periphery right or left. The objects depicted in the centre of the two retinae, or other functionally identical places, are blended into a uniform cerebral impression, or binocular vision. However, to a certain extent the brain is also capable of fusing pictures which do not fall upon geometrically identical places of the retinae, as is proved by the ability to observe near objects and by the perception of depth. This is a purely psychic process, known as the fusion ability. Worth and others have made the following observations on the development of binocular vision: the pupillary reaction present in earliest

infancy, and the "fixation" reflex of the ophthalmoscope, on the part of an infant only a few hours old, show a normal act of the macula lutea of each retina from the moment of birth. Also, the uniform movement of the eyes upward or downward proves the association of both eyes for vertical movements.

The development of the association of the two eyes in horizontal movements, which is of the utmost importance for the binocular process of vision, has occurred in the fifth or sixth week, with sufficient exactness to reflect pictures of an object upon symmetrical points of each cornea. While one eye, however, engages the ophthalmoscope, the other is sometimes deflected inwardly to a slight extent or, in rare cases, outwardly. The coördination of both eyes in the horizontal plane, and their fusing ability, are still so uncertain in the first months of life that a gastric or other disturbance may cause the deflection of an eye, or an inward squint of both eyes, for a few seconds or longer.

After about the first year, the eyes exert themselves to a greater degree to effect binocular vision, so that the power of fusion is normally developed before the end of the sixth year.

The ability to fully appreciate space is acquired in the third year, based upon distinct impressions retained in the memory. Increased experience and perception serve to develop exact vision, such as measurement by the eye, perception of distance, estimation of depth, and the development of visual memory. Boys usually show greater aptitude in developing these characteristics than girls.

It may also happen that this visual experience is lost after having been possessed. This may be a purely psychic disturbance, as when a child becomes temporarily blind through blepharospasm or, for a longer time, from cataract. As is the case in those congenitally blind who have been successfully operated upon, vision must be reacquired after the cataract or blepharospasm has been removed.

As to the color-sense of infants, it has so far been found that the new-born infant does not have any clear color perception, but merely general impressions of light, dark, and color. Preyer observed in his infant, when one month old, that color caused pleasure. In the second year the color-sense was fully developed, and in the third year the colors could be correctly named.

For fifteen days W. A. Nagel studied the color-sense of a child two years and four months old. The child learned immediately not only the red of the spectrum, but also other tints of red, including rose, purple, and sometimes orange. Green was rapidly distinguished and retained; violet and black were recognized a few days later, while gray and blue required a little longer. The name for blue had been forgotten in two days, but the color itself was but rarely mistaken for others.

K. L. Schaefer states that the ability to discern the principal colors—red, yellow, green, blue, and violet—sets in simultaneously at a certain stage of development. This depends upon the examination itself or other accidental causes, such as a predilection for a certain color. For instance, if a certain color test is used at one time, and a different test at another time, in order to test the accuracy of the color perception, the response to a particular color is prompt. Blue and red are usual favorites, and saturated tints give more pleasure than pale ones.

The absolute perception of colors requires first the optical distinction, then the correct name, and finally a satisfactory memory.

The ability or inability to distinguish colors is therefore congenital, as they are present as soon as the eye develops, the brain is capable of perception, and the necessary nerve connections are formed. A congenitally blind eye which has been successfully operated upon can immediately distinguish colors like a normal eye; indeed, various qualities of light have already been recognized before, although not to the same extent.

The ability or inability to distinguish colors is no test of congenital intelligence. F. Warburg, however, found the ability to name colors a good guide in determining whether the intelligence of a child is so restricted as to require instruction in special classes. Weak-minded children acquire color distinction very late, or not at all. Mental inferiority may therefore be assumed to exist when a child, upon entering school, cannot name black and red correctly, while the ability of a six- or seven-year-old child to distinguish brown, gray, etc., correctly is proof of superior intelligence.

Kannegiesser states that pupils attending his special school, whose color-sense was markedly defective, could not be promoted to higher grades at the end of the year.

Gelpke found that a large percentage of demented children, in all probability, had the correct color-sense, but were unable to name the colors correctly. Girls usually have a better color-sense than boys, probably because their dresses and other articles belonging to them display more diversity of colors.

E. Kahn states that the color-sense is not fully developed in the majority of children during the first school years; usually, the names for pink, orange, and violet are missing.

The refraction of the eyes in the new-born is nearly always hypermetropic. This usually amounts to 2 or 3 D, and is not caused so much by a short optic axis as by increased curvature of the cornea and increased bulging of the lens. It is often accompanied by regular astigmatism, with increased refraction of the vertical meridian. Elschuig states that myopic refraction of 4 to 7 D is often found with the skia-

scope in from two hours to two days after birth, chiefly in infants whose eyes display active movements shortly after birth. Instillation of atropin causes the myopia to give way to a slightly hypermetropic state of refraction. In some infants, especially those with convergent and active upward and downward ocular movements, the refraction varied, and there was transient myopia. Here, again, paralysis of accommodation through atropin showed hypermetropic or approximately emmetropic refraction. In some cases there is originally hypermetropic or approximately emmetropic refraction; these are usually children with very large pupils, and those whose eyes are almost motionless when lying down.

Spasm of accommodation, therefore, renders many infants myopic; paralyzing the accommodation with atropin will make the conditions disappear.

Spasm of accommodation, which in many cases is quite constant, and in others variable during examination, is attributable to unsuitable and involuntary innervation following the first attempts at vision, and an abnormal reaction of the infantile eye to the first rays of light.

Congenital myopia in the true sense has never been observed. The majority of juvenile eyes become emmetropic in the course of years, or "typically normal," according to Donders. The ideal condition of a pencil of rays starting from an illuminative point on the retina, and then becoming parallel after passing through the dioptric system, does not often occur, according to Straub. On the other hand, this condition is partly the result of an adaptation by the lens to anomalies which exist in the corneal curvature or in the length of the optic axis.

Aside from the fact that hypermetropic eyes are of shorter axial length than myopic eyes, they are further distinguished by a difference in the principal focal distance of the lens. The first factor causes the refractive error, the second attempts to correct it. Accordingly, the refractive power of the lens is greater in hypermetropic than in emmetropic and myopic eyes.

Psychic weeping is completely absent in the new-born, and reflex weeping occurs only to a small extent. However, irritation of the mucous membrane of the nose causes tears, showing that the lachrymal glands are already capable of secretion. Sommer observed absence of both forms of weeping in an otherwise normal child of two and a half years; he also states that psychic weeping occurs later in weak children than in those well developed.

III. EXAMINATION OF CHILDREN WITH OCULAR AFFECTIONS

THE examination is not restricted to the eyes, competent treatment of the latter requiring a thorough examination of the entire constitution. Pathological changes of the eye often point to organic or constitutional affections, and in other cases the eyes are secondarily affected. With each patient it is important that the eyes should be systematically examined. But functional tests as to central vision, refraction, accommodation, light-sense, color-sense, field of vision, space-sense, movements of the eyes and positional anomalies, binocular vision, are not easily applied with infants, and in most cases they can be effected only after a certain degree of intelligence has developed.

The child should not be frightened, and with excitable children the examination should be made with more than ordinary care. There should be no intense illumination, as for instance from a bright window; but some place in the room should be selected where the illumination is evenly diffuse. The child should not be touched or held while the anterior sections of the eye are being examined, but in case of need very gentle traction may be made on the upper lid with the finger. A watch or some other bright object may serve to divert the attention. If there is photophobia, half the eye should be screened.

The bulbar conjunctiva and the anterior section of the sclera may be seen by drawing the lid up and down from the globe, and having the patient look downward, upward and laterally. The inferior tarsal conjunctiva and the inferior fornix are inspected by drawing the lower lid downward with the thumb placed near the palpebral margin, while the patient looks upward.

Inspection of the superior tarsal conjunctiva and the superior fornix requires eversion of the upper lid, which in little children is effected as follows: the thumbs of the right and left hands are placed on the anterior surface of the upper and lower lids, which are now pulled apart. By exerting pressure against the orbital margin—not the globe—the lids are turned over, exposing the tarsal conjunctiva in its entire extent.

In order to see the fornix, it is often necessary to turn the lid over twice. To do this, the thumb, or a small glass rod, is held upon the everted margin of the lid (upper margin of the tarsus), and both index-fingers are placed flat upon the palpebral margin, after which the lid is turned over again. The upper fold may be exposed as follows: the upper lid having been everted as described above, it is held with the left thumb. The patient should look down as far as possible. The right thumb is placed flat upon the globe, which is covered by the lower

lid, and gently but powerfully pushed backward into the orbit. The everted lid is slightly drawn upward with the left thumb, the patient being instructed to look continually downward. This will cause the upper folds to protrude like a ridge, allowing inspection as convenient.

As these manipulations are somewhat painful, it may be advisable to instil a 2 to 3 per cent. solution of alypin or 2 to 3 per cent. cocaine and suprarenin.

The eversion of the upper lid may prove impossible if, for instance, the tarsus is deformed or very stiff, if the palpebral margin is rounding, or is deprived of the ciliae, and especially if the conjunctiva is considerably shortened. The right thumb in such a case is placed on the surface of the lid, as just described, and the palpebral margin is firmly pushed from below upward with the left index-finger or the thumb.

Inspection of the anterior sections of the eye may be made difficult by œdema of the lid, and still more by blepharospasm.

If local anæsthesia is desirable, as, for instance, in constriction of the lids, cocaine suprarenin is instilled ten minutes before the examination. This solution is 3 per cent. cocaine muriat., or salicylic. 5.0, to which are added five drops of suprarenin synthetic., or paranephrin 1.0 : 1000.0. The fluid is made pleasantly warm, so as to prevent its being diluted or expelled by crying. It is instilled from the outer canthus rather than at the centre of the eye, so as not to frighten the child unduly (Fig. 12).

Sometimes, though, children resist the ocular inspection, or instillation of cocaine suprarenin may be impossible, as in infants recently or prematurely born, or in blepharospasm due to phlyctenular inflammation of the eye, etc. In such cases the physician, having seated himself with his back toward a window, and with his feet resting on a footstool, takes the head of the infant between his knees, which are covered with a towel. The nurse, sitting opposite him, holds the child in the dorsal position so that its legs are underneath her arms, and with her hands she draws down the child's hands.

The lids are cleansed, cocaine suprarenin is instilled if indicated, and the lids are carefully and gently drawn apart so that the surface of the globe can be freely inspected. The palpebral conjunctiva, however, is not pressed forward, lest it should cover the cornea.

If satisfactory inspection cannot be obtained in this way, or if digital pressure upon the globe is dangerous, because of the possible perforation of deep and partly healed corneal ulcers, the plate of a Desmarres blepharostat is carefully inserted behind the upper lid, upward and forward away from the globe. If it is not possible by drawing down the lower lid with the disengaged hand to see the field clearly, then another blepharostat is inserted behind the lower lid. For this purpose, I like to use blunt double hooks of sufficient size, as they exert less pres-

sure than the specula. The insertion of a lid speculum is not advisable, as there is danger of perforation of the cornea, and it also increases irritation. General anæsthesia is indicated for frightened or unruly children, ethyl bromide being sufficient, as only a short narcosis is required. This should be administered, however, by an expert, and the effect constantly watched, so that the pupil, for instance, is never allowed to become dilated. This will also facilitate the inspection of the cornea, especially if a simultaneous instillation of fluorescein has revealed indistinct epithelial desquamation and marginal ulcers. This method of

FIG. 12.



Instillation.

examination is therefore indicated in patients with deep corneal ulcers, especially if the globe persistently rolls upward, and also in serious injuries to the eyes. The same course is adopted when changing bandages, if necessary, especially after serious operations, like the removal of cataract, etc.

All these manipulations are more easily carried out with the patient in the recumbent posture.

If the presence of infectious material, especially of gonorrhœal secretion, behind the œdematous or contracted eyelids is suspected, or if there is any other danger of infection, the operator will do well to protect his eyes with large watch-glass spectacles before everting the lids. In-

fection from secretions of any kind, especially in affections of the lids, lachrymal organs, and conjunctiva, should be guarded against by disinfecting the hands carefully, both before and after the examination.

As soon as the conjunctiva is exposed, its surface is gently cleansed with a sterile cotton carrier, in order to facilitate a more careful inspection.

General anæsthesia is contra-indicated in children with enlarged thymus or a lymphatico-chlorotic constitution, as these conditions are characterized by a pathological disposition to sudden death. They are shown by follicular hypertrophy of the base of the tongue, especially by the simultaneous presence of hyperplasia of the lymphatic tonsils, of the follicles of the posterior faucial wall, of the faucial tonsils, of the lymph-glands, and of the spleen. The lymphatic diathesis is often associated with hypoplasia of the arterial system and the genitals. In older children pronounced hypertrophy of the lingual tonsils can be noticed without difficulty by depressing the tongue with a spatula or by using the laryngoscope. The hypertrophy sometimes appears in the shape of multiple eminences, sometimes as a sessile or pedicled tumor the size of half a walnut; in other cases only one-half of the lingual tonsil may be hypertrophic. This condition may exist without giving rise to any symptoms, or there may be various disturbances from mechanical causes,—as, for instance, the so-called globus hystericus,—or also from reflex neuroses. Examination of the structures mentioned is just as important as examination of the heart.

IV. AFFECTIONS OF THE EYELIDS

THESE affections may involve the lids alone, or they may be caused by pathological changes in the conjunctiva and the drainage portions of the lachrymal apparatus. The conjunctiva forms the posterior layer of the lid. The horseshoe-shaped part of the lid fissure at the inner canthus contains the puncta lacrimalia on the posterior margin of the lids, at the crest of the lachrymal papilla. Affections of the globe and orbit may likewise cause changes of the lids, as they also cause paralysis and spasms of the facial, trigeminus, and sympathetic nerves, and changes in adjacent parts of the face.

It will thus be seen that many points are to be taken into consideration. The two eyes should be constantly compared, in order to detect any shortening of the lids, either congenital or acquired, any thickening of tissue layers and parts of the lids from inflammation, inflammatory or pure œdema or lymphatic congestion, thickening of the tarsus, swelling of the tarsal conjunctiva, or new formations of skin or cellular tissue. Adhesions of the eyeball may be complicated by bulging of the lids, accumulation in the lachrymal sac, inflammation or tumor of the lachrymal glands, osteophytes or enlargement of the orbital bones, enlargement or bulging of the eyeball, orbital tumors, tenonitis, etc.

Retraction and flattening of the lids may be caused by enophthalmus, bulbar phthisis, or defective bones of the orbital margin. Special attention should be paid to anomalous position, entropion, ectropion, or changes in the color, such as hyperæmia, vascular dilatation, hemorrhages, or pigmentation.

The physiological condition of the lid margins should be examined for pathological changes, in the shape of deposits of epidermal or fat scales, crusts (desiccated secretion of the conjunctiva or pathological secretion of the palpebral glands), scabs (dried-up ulcerous secretions) on the epidermis of the external palpebral margin and ciliæ, nodules, vesicles, cysts, new formations, ulcers, scars, or injections. The physician should also note any rounding of the palpebral edges from chronic inflammation of their margins and of the conjunctiva, any contraction of the hair area, displacement and loss of the ciliæ, madarosis (Plate I, Fig. 4), distichiasis, or trichiasis; any changes in the puncta lacrimalia, such as absence of one or both of them, cicatricial adhesions, etc. Nor should changes in the opening of the lids (in the palpebral fissure) and their contents, such as ankyloblepharon, blepharophimosis, lagophthalmos, ptosis and true ptosis caused by paralysis (Fig. 1), pseudoptosis with hanging lid in trachoma (Fig. 13), or symblepharon, etc., be overlooked.

Other symptoms are absent or defective closure of the lid, with or without lachrymation, which occurs in spasm of the oculomotor nerve, paralysis of the facial nerve, exophthalmos (Fig. 14), and ectropion; im-



FIG. 13.



FIG. 14.



FIG. 15.

FIG. 16. FIG. 17. FIG. 18. FIG. 19. FIG. 20. FIG. 21. FIG. 22. FIG. 23. FIG. 24. FIG. 25. FIG. 26. FIG. 27. FIG. 28. FIG. 29. FIG. 30. FIG. 31. FIG. 32. FIG. 33. FIG. 34. FIG. 35. FIG. 36. FIG. 37. FIG. 38. FIG. 39. FIG. 40. FIG. 41. FIG. 42. FIG. 43. FIG. 44. FIG. 45. FIG. 46. FIG. 47. FIG. 48. FIG. 49. FIG. 50. FIG. 51. FIG. 52. FIG. 53. FIG. 54. FIG. 55. FIG. 56. FIG. 57. FIG. 58. FIG. 59. FIG. 60. FIG. 61. FIG. 62. FIG. 63. FIG. 64. FIG. 65. FIG. 66. FIG. 67. FIG. 68. FIG. 69. FIG. 70. FIG. 71. FIG. 72. FIG. 73. FIG. 74. FIG. 75. FIG. 76. FIG. 77. FIG. 78. FIG. 79. FIG. 80. FIG. 81. FIG. 82. FIG. 83. FIG. 84. FIG. 85. FIG. 86. FIG. 87. FIG. 88. FIG. 89. FIG. 90. FIG. 91. FIG. 92. FIG. 93. FIG. 94. FIG. 95. FIG. 96. FIG. 97. FIG. 98. FIG. 99. FIG. 100.

ularis (facialis); Basedow's (Graves's) disease; absent or imperfect involuntary reflex movements of the lids, or their retarded or rare occurrence (Stellwag's symptom); absent or diminished associated movements of the upper lid with downward vision, the lid at first failing to follow the movement of the bulb, as if retained by a spasm, and later executing some feeble, fitful downward movements (v. Graefe's symptom).

A more or less pronounced gaping of the palpebral fissures may occur in one or both eyes, independently of bulbar protrusion, and may simulate exophthalmos. Kocher mentioned retraction of the upper lids, when some object is rapidly moved up and down in front of the eyes.

A tactile and thorough examination of the lids, with comparison of both eyes, is indispensable. In the presence of tumors and inflammatory foci the movability of the palpebral skin is an important symptom. A giving way to pressure may indicate angioma or a meningocele. Fluctuation, elevation of temperature, grating and crepitating sensations should be noted in injuries to the eye. Pulse vibrations, lowered sensation or anæsthesia of the palpebral skin, such as occur in herpes zoster, and susceptibility to pain on touching the eyelids, should also be looked for.

Open wounds of the lids, superficially visible traumatic canals, fistulæ of the lachrymal gland or sac, and caries of the bones, require the gentle introduction of sounds. Constriction of the puncta lacrimalia and canaliculi demands similar treatment.

1. SPONTANEOUS (NON-TRAUMATIC) HEMORRHAGE AND CIRCULATORY DISEASE OF THE LIDS

Petechiæ are of particular importance in leukæmia, pseudoleukæmia, purpura hæmorrhagica, and morbus maculosus. Small, circumscribed hemorrhages of the lids occur in grave cases of scarlet fever, and macular palpebral blood extravasations in the so-called septic changes of the retina. The hemorrhagic transudation of the lids in infantile scurvy (Barlow's disease) disappears rapidly as soon as over-sterilized milk is replaced by pure raw milk and a fruit diet, such as orange juice and finely minced apple. Hummelsheim, who noted exophthalmos with hemorrhagic suffusion as the only early symptoms, states that they disappear with the later symptoms of distinct pain below the epiphyseal borders of the femur, etc. In pertussis, hemorrhage of the lids may occur by bursting of blood-vessels. This is usually accompanied by hemorrhage of the conjunctiva (Plate III, Fig. 4), which ceases when the violent coughing is relieved. In hemophilia, v. Michel observed in a child a hemorrhage which extended to the lid, forehead, and the affected side of the skull, following a slight blow in the region of the eyebrow. As a rule, these hemorrhages disappear after the skin has passed through the usual discolorations. However, a cooling band-

age such as a three per cent. boric acid solution, or lead water with equal parts of distilled water, may be applied.

Compresses and puncture are required only in extensive hemorrhages.

Active hyperæmia and inflammatory swelling of one or both lids, of varying degree and extent, and accompanied by increased tension, resistance, and local temperature, as well as susceptibility to pressure, occur in inflammations due to furuncles and to erysipelas, or in infections such as the various forms of acne. This is also the case in folliculitis and perifolliculitis of the meibomian glands (hordeolum internum, Plate I, Fig. 6). They are also very often concomitant manifestations of gonorrhœal and diphtheritic conjunctivitis, iridocyclitis or glaucoma. Furthermore, phlegmons of the orbital cellular tissue are observed in panophthalmia, in suppurative affections of the lachrymal sac and of the accessory sinuses of the nose, in periostitis of the orbital margin and the superior maxilla. In parotitis the external parts of the upper lid are likewise favorite loci. If both lids are involved, the upper lid is usually more enlarged than the lower; in acute dacryocystitis (Plate II, Fig. 5) and empyema of the frontal and maxillary sinuses the neighboring medial part of the lids is usually attacked; in acute dacryoadenitis, the central part. Both the hyperæmia and the swelling of the lids usually disappear with the cure of the underlying affection, but swelling and relaxation of the upper lids with pseudoptosis may persist for some time.

Engorgement hyperæmia of the lids is a bluish-red, œdematous swelling, which usually involves the tarsal and scleral conjunctivæ, and may be caused bilaterally by cyanosis, cardiac insufficiency, or by Asiatic cholera. Unilaterally it is due to circulatory obstruction of the jugular vein and the sinus cavernosus, as, for instance, in the presence of thrombosis, thrombophlebitis, orbital tumors, pressure of swollen lymph-glands upon the common jugular vein, and persistent reflex spasm of the musculus orbicularis.

Treatment consists in the removal of the underlying cause. Should thickening of the upper lid persist, resembling elephantiasis, superficial painting with tincture of iodine, repeated if necessary, may cause its disappearance; otherwise partial excision may have to be resorted to.

The following conditions are of general diagnostic importance: œdema of the lids, with conjunctival irritation and increased lachrymation, due to arsenic and iodine poisoning; erythema of the lids in febrile diseases, œdema in myxœdema, which usually occurs in the lower lids; œdema of the lids in vasomotor migraine; the cushion-like swelling which, according to Saenger-Ludeck, occurs early in Basedow's (Graves's) disease; the acute circumscribed recurrent œdema of the lids setting

in spontaneously and accompanied by œdema in other localities (Quincke's disease). Of equal importance are transient œdema in influenza, trypanosomiasis, chronic nephritis, beri-beri, and in scarlet fever; also the pale, diaphanous œdema in trichinosis which occurs at the end of the first week with chemosis of the conjunctivæ after an attack of diarrhœa. Then there is facial œdema with muscular pain; persistent œdema due to lymphatic stasis, occurring unilaterally or bilaterally and either in one or both lids, although usually the upper one is affected. This often involves parts of the face and causes nasal affections, such as chronic rhinitis, polypous proliferations, and erysipelatous inflammations.

œdema of the lids should not be confused with leukæmic or pseudo-leukæmic small tumors and palpebral infiltrations, which often attack the four eyelids symmetrically and do not extend to the subconjunctival tissue, as do lymphomatous tumors. Besides, the skin can be raised in these growths, which is not the case in œdema.

The irritation of the conjunctiva in scarlet fever is often caused by direct infection. In the stage of desquamation it may lead to œdematous catarrh of the conjunctiva, with subsequent ulcers of the corneal margin.

The exanthemata of the lid in measles, rubeola, and scarlet fever are often accompanied by excoriations of the skin.

The treatment for this condition is careful inunction of the lids with a thin layer of a 3 per cent. boric acid ointment, or we may use ungt. hydrarg. præcip. alb. pultiform. (0.1 lanolin, vaselin. Amer. ãã 5.0).

2. VARIOLA OF THE LIDS

The involvement of the lids by smallpox is in proportion to the eruption on the face. Hemorrhages in the lids usually accompany hemorrhagic variola. If the eruptions are numerous, the œdema may close both lid fissures. This condition is often accompanied by burning pain. Although the œdema itself is not dangerous, it interferes with the examination of the eyes, which should be done daily, as there is frequently metastatic involvement of the conjunctiva, cornea, and globe. In difficult cases treatment by an ophthalmologist is therefore indispensable.

The diffuse pustules, which are located at the upper palpebral margins more often than at the lower ones, are apt to spread to the palpebral and scleral conjunctivæ. As these pustules burst, they form crusts agglutinating the palpebral margins and cilia, the latter falling out. This gives rise to ulcerations, similar in appearance to those of diphtheria, which, owing to continuous invasion by lachrymal secretion, heal with difficulty. The entire palpebral margin may thus be changed

to a single, slightly hemorrhagic, ulcerous area, which in turn may lead to depressions and more or less extensive adhesions of the palpebral margin. Destruction of the hair-follicles, permanent loss or malposition of the cilia, ulceration of the hair bulbs, chalazia, hordeola, and occlusion of the meibomian glands may be the result. Cicatricial ectropion, especially of the lower lids, may result from confluent smallpox, or entropion from lesions and retraction of the inner edge of the lid may follow cicatricial deformities, with thickening of the entire margin. Red spots sometimes persist at the margins, becoming more hyperæmic with each rise of temperature. The cornea is in particular danger, as mentioned in the chapter under that heading.

Differential Diagnosis.—Variola can be confused with papulous measles only at the very onset of the eruption, the former being indicated by the burning pain, and marked œdema of the lids.

The following figures relating to vaccination are deserving of consideration. In Bohemia there are two hundred and fifty-three blind people who owe their affliction to smallpox; in Bavaria, which is of equal size, there are only twenty-two, twenty of whom contracted the disease before repeated vaccination was introduced. In Sweden, with a population of five million, there is no case of blindness from smallpox, and this decrease did not become evident until after the introduction of repeated vaccination.

Treatment.—Inasmuch as immunity is only for a term of years, E. Levy suggests first of all that physicians and nurses in attendance upon patients suffering from smallpox should be immediately vaccinated or revaccinated. It has often occurred that other patients, as well as physicians and nurses, have been infected in hospitals. If, in spite of this precaution, a freshly-vaccinated individual should contract the disease, it can be either aborted or will run a mild course, since the vaccination has already entered the suppurative stage before variola has fully developed, and therefore the more satisfactory is the result.

The eruption itself is perhaps lessened by covering the face, with the exception of the eyes, nose, and mouth, with an aseptic slightly warmed linen bandage moistened with a mixture of equal parts of vinegar and cold water. On this small pieces of ice can be laid. But if the eruption has already appeared, we can perhaps confine the suppuration within the narrowest limits possible by moistening the bandages with equal parts of a very weak solution of muriatic and nitric acids in distilled water. A crust soon forms which is covered with a thin, neutral layer of borated lint, until the scabs fall off. The application is renewed every morning and evening, the eyes, nose, and mouth remaining free. This arrests scab formation at the lids, agglutination of the cilia and retention of secretion in the angles. The most suitable ointments are

boric acid with vaseline-lanolin (boric acid 2.0, vaseline-lanolin $\bar{a}\bar{a}$ 25.0, M. F. ungt.) or diachylon ointment.

The ointment is removed slowly and carefully and from time to time a cool bandage moistened with diluted chlorine water is substituted. Before each fresh application, the lids and their margins are carefully and gently cleansed with sublimate (1 to 5000) and then dried. After desquamation of the scabs, powdered starch is applied where the process of healing has begun. The whole procedure should be clearly shown to the attending nurse.

The light in the room should be subdued without, however, interfering with ordinary ventilation.

FIG. 16.



Taermophore.

FIG. 17.



Japanese warming box.

Rubbing the eyes to relieve itching, especially when the crusts dry, increases the conjunctival hyperæmia and induces penetration of the decomposed skin into the conjunctival sac. It should therefore be avoided.

Photophobia, blepharospasm, and the desire to rub the eyes are relieved by instillation of a 2 per cent. cocaine solution, repeated several times a day. With intractable patients and children, the hands and arms are confined by suitable bandages or by cardboard splints. Adhesions of the palpebral fissure are prevented by pulling the lower lid away frequently, and separating the agglutinated parts gently with an aseptic, thin, blunt glass rod, or a conical sound of larger calibre.

Sties are ripened by applying very weak on the bandages a cataplasm, as suggested by Langlebert.

Large hordeoli, which often form a circumscribed thickened projection on the lid, are best removed by galvanocautery, using a finely-pointed instrument either at the lid or tarsal conjunctiva, as indicated (Plate XXI, Fig. 10). But this operation should be delayed, if possible,

until the eruption has subsided, since œdema of the lids may interfere considerably with the work.

The œdema itself does not call for any special treatment. But should it persist after the disease has been cured, without changes in the cartilage, the application of dry heat is indicated. It may be made either by an electro-thermophor (Fig. 16), or a warming box (Fig. 17), which is fastened without pressure over one or both eyes with gauze. The box is enveloped in cotton, so as to prevent blistering. Careful painting with tincture of iodine has a favorable effect. This should be repeated once or twice after the lapse of eight or ten days.

Smallpox ulcers, which appear later, are best cicatrized by careful superficial galvanocautery, or careful touching with chlorinated silver nitrate, fused to a fine sound.

Faulty position of the cilia and marginal erosions, which may cause permanent irritation of the conjunctiva, lachrymation and corneal ulcers, should be corrected by drawing out the hairs with ciliary forceps (Plate XXI, Fig. 8) or, preferably, by electrolytic destruction of the hair-follicle. For the same reasons eversion or inversion of the lid is corrected by massage or appropriate bandages. If these measures prove unsuccessful, the services of a specialist should be procured; in atypical cases this should be done from the first.

3. VACCINE INFECTION OF THE LIDS

An auto-infection of the eye by vaccine pustules or an infection of a non-vaccinated person, or of one who is no longer immune to the vaccine poison, may occur in the form of a vaccine blepharitis (Plate II, Fig. 3).

Three or four days after the infection a little vesicle, with a central indentation, or more frequently a small superficial pustule appears at the intermarginal part of the lid and adjacent palpebral conjunctiva. This soon changes to a typical ulcer. It usually disappears, either without leaving a trace, or, in rarer cases, there remains only a slight scar with a loss of cilia over the ulcerated base. The pustules often begin with an inflammatory œdema of the lids, which interferes with the opening of the eyes. The superficial ulcer is of circular shape, covered by a whitish layer resembling that of diphtheria, which can be detached in large flakes. The surrounding area is deeply infiltrated. Gradual extension of the ulcers and increasing œdema, which may spread to the malar and temporal regions, lead to chemotic swelling of the bulbar conjunctiva. Swelling and increased pressure of the preauricular lymph-glands on the affected side cause considerable interference with the general condition, sometimes accompanied by increased temperature, lassitude, depression, and anorexia. Other pustules and

ulcers may form by direct contact of opposite parts of the upper and lower intermarginal lids, but these rarely acquire the size of the original ulcer. This is also true of ulcers which spread from these points to the cul-de-sac and even to the bulbar conjunctiva.

Superficial marginal ulcers of the cornea, occurring at the climax of the disease, heal rapidly. On the other hand, diffuse central and deep infiltrations, in the form of striæ and hooks, which impart to the corneal surface a stippled appearance, sometimes causing retinitis and deposits on the posterior surface of the cornea, will never completely disappear.

Typical vaccine pustules appear less frequently than vaccine ulcers. Women are more likely to be affected than men, because of their closer relation to children.

The *diagnosis* is indicated by the history, the negative bacterial findings, the rapid course of the disease, and the high degree of contagion.

In the differential diagnosis diphtherial infiltrations, erosions of the palpebral margin, syphilitic and tuberculous ulcers, and suppurative carcinoma must be considered.

In so-called vaccine "generalisata" the lids may also be involved. This usually occurs in eczematous children, so that the eruption is probably not hæmatogenous, but due to inoculated eczema or vaccine eczema (Voight).

The *prognosis* in pure lid affections is favorable, especially if due to auto-infection. Tertsch, however, observed extensive necrosis of the lids and margins in a secondary infection with palpebral vaccine.

Treatment.—The relatives of vaccinated patients should be acquainted with the ocular danger involved in uncleanness, and the danger from fingers soiled with secretion. Infection has been known to occur by washing a child's face with a towel or sponge which had been used on the vaccinated arm of another child. Children who suffer, or have suffered, from blepharitis and other ocular affections should be particularly guarded from infection. Owing to the possibility of contagion, freshly vaccinated children should sleep alone. Crust-formation by thermo- or galvanocautery or incandescent platinum needle, such as is sometimes used for bacteriological purposes, should be attempted only in the very first stage of the palpebral affection. By following this rule, the formation of fresh marginal pustules and lingering central corneal affections will often be prevented.

Applications of tepid chlorine water will favor the desquamation of the necrotic parts; they are best applied to the lid on sterilized absorbent gauze, three or four times daily for fifteen or twenty minutes at a time.

A cooling ointment spread on lint or applied as a bandage gives a sensation of relief.

For treatment of corneal affections, see chapter on "Cornea."

4. ERYSIPELAS OF THE LIDS

This affection is due to excoriations and trivial lesions of the lids, or to the spreading of a facial erysipelas, as, for instance, in variola. Lymphogenic osseous foci of suppuration develop comparatively often in the mucous membrane of the nose or in the accessory nasal sinuses. In most cases the lids are uniformly hyperæmic, accompanied by such pronounced œdema that the eyes can be opened only with difficulty. Vesicular or bulbous erysipelas is not rare. Involvement of the deep parts may cause palpebral abscess (Plate II, Fig. 1), as well as suppuration of the lachrymal sac, if the inner canthus is preëminently involved. Gangrenous erysipelas and mixed forms of erysipelas and phlegmons of the lids are rare.

An approaching abscess formation may be recognized by increased inflammatory œdema, hardness of the deeper parts, swelling of the pre-auricular and submaxillary lymph-glands, elevation of temperature, and enervation.

The chief point in differential diagnosis between abscess formation and a phlegmon of the lachrymal sac is that in the latter the region between the inner canthus and the bridge of the nose is the one chiefly involved. Moreover, it does not show the raised inflammatory area of erysipelas nor its characteristic progression. It may be impossible sometimes to make a clear distinction, but, as the treatment is practically the same, no particular importance attaches to this possibility.

Palpation of the region of the lachrymal sac, which is important in diagnosing acute dacryocystitis, requires great care, since the sac may have perforated posteriorly, and any exaggerated pressure upon it will favor the spreading of the suppuration to the orbit.

Herpes zoster, too, may resemble erysipelas, except that the former is sharply defined by the medial line of the face. The neuralgic manifestations and paræsthesia of the skin are absent in erysipelas.

So-called habitual erysipelas of the face and the resulting hard, tough, connective-tissue thickening of the lids (elephantiasis) are due to occlusion and dilatation of the lymphatic vessels. The causes are chronic eczema, adenoid vegetations, faucial catarrh, chronic affections of the lachrymal ducts and nasal sinuses, or catarrh and empyema of the superior maxilla. The lower lids are sometimes so swollen and shapeless as to hang down like wrinkled, diaphanous bags, resembling alabaster. The eyes seem to be reduced in size, because of the constriction of the palpebral fissures. A thickening and widening at the root of the nose may simulate epicanthus.

The *treatment* consists, in the first place, in the removal of eczematous tears and abrasions in the anterior and posterior angles of the nostrils. This can be done by carefully painting them with a 2 per cent.

solution of silver nitrate, preceded, if necessary, by rinsing the nose with a 3 per cent. tepid solution of boric acid. Extensive inunction with ichthyol or precipitated white ointment (Hg praec. alb. 0.5, lanolin-vaseline 25.0 āā), followed by a 2 per cent. carbolic vapor bandage or by alcohol-sublimate with gutta-percha, will arrest extension to the deeper parts of the lid and to the orbit. If the infiltrate area has softened, as indicated by the presence of fluctuation, early incision should be made, owing to the impending danger to the orbital and intracranial structures. On the lids the incision should run horizontally, but over the lachrymal sac it should be directly obliquely from inward and upward, downward and outward. The vapor bandage is continued until the ulceration is arrested. If perforation into the subtarsal and conjunctival tissues is imminent, another incision should be made from the side of the conjunctiva. This should have a vertical direction, if possible, in order to avoid lesion of the meibomian glands. Should the destruction of the skin of the lid be very extensive, the treatment described on p. 106 may have to be considered.

A complete cure is essential to prevent relapses, as any subsequent softening by massage or otherwise of parts of the lids and face which have been hardened is difficult in proportion to the time that the inflammation has existed.

5. ANTHRAX INFECTION OF THE LIDS

This infection sets in with oedema, induration, and hyperaemia of the skin. It may spread rapidly to the scalp and face, causing violent headache and benumbing of the senses. A typical ulcer rapidly develops by enlargement of the pustule, which at first is small, but slightly painful, and surrounded by a red areola. As the disease progresses, the centre of the ulcer is indented and covered by gangrenous crusts. This indentation may be surrounded by a reddish-violet, infiltrated areola and a number of vesicles containing a reddish-black fluid. Schmitt observed in a six-year-old child malignant oedema due to streptococcus and pneumococcus infection, which terminated in a cure. Both lids are often destroyed. Or the margin of the lid may alone escape infection owing to its better nourishment. After desquamation of the gangrenous parts, eversion of the lids and lagophthalmos may occur, with the sequelæ of those conditions in the conjunctiva and cornea.

Sulzer observed a case due to the thrust of a cow's horn, and Antonelli of Naples considers mosquitoes a frequent causative agent.

It is an important point in the diagnosis that anthrax may cause erysipeloid swelling and reddening of the skin. In dubious cases, therefore, the blood and oedema have to be microscopically and bacteriologically examined.

The *prognosis* of anthrax of the lids is serious, as the anthrax bacilli may enter the circulation and thus endanger life. It is, however, more favorable than when anthrax attacks other parts of the body. But when the bacilli are once present in the circulation, the course of the disease is usually fatal.

Formerly the *treatment* consisted in the destruction of the pustules by thermocautery or by a deep circular incision penetrating into the subcutaneous cellular and muscular tissues. In advanced stages a very extensive and deep incision was made, parallel to the palpebral margin, with cauterization of the traumatic surface with caustic potash, and subsequent application of heat in any shape most agreeable to the patient. But this method invites infection of the blood, and promptly leads to metastases to the internal organs. For this reason, v. Bramann advises rest in bed, an antiseptic bandage over the eyes, nutritious diet and concentrated alcohol, with inunction of the entire infected area with gray ointment. Axenfeld, Ménétries, and Ceunet give the preference to anthrax serum, and Morax to injections of potassium iodide into the region surrounding the carbuncle.

After the pustule has been completely evacuated, temporary closure of the lid fissure may prevent exaggerated shortening of the lid in cases where a large area is involved. This operation is performed by freshening up and suturing the palpebral margins at several approximately equidistant places and covering the ulcer with epidermal flaps, one slightly overlapping the other.

Characteristic cicatricial deformities and high degrees of ectropion frequently occur at a later period. For this reason, even apparently well-healed cases require protective spectacles, especially if the patients are exposed to dust. In order to prevent contraction of the scar, so far as is possible, boric ointment should be applied to the lid, both morning and evening. Should the lids, nevertheless, become shortened and everted, blepharoplastic surgery may be necessary, but nothing should be done until the deformities have assumed a definite form, as otherwise operation may result in failure.

6. GANGRENE OF THE LIDS

Metastatic gangrene in septic pyæmia and ulcerous endocarditis, which has always been regarded as a harbinger of death, has, among its various causes, a mycotic embolism of the arterioles emanating from the arcus tarsei. The metastases often involve the four lids. The forms of lid gangrene, described as due to varicella, measles, German measles, scarlatina, and typhoid (Fig. 18), and also the symmetrical gangrene known as "noma" of the lids, are probably all due to septic embolism. The neurotic necrosis of ophthalmic herpes zoster, and the

ectogenic necrosis which occurs in varicellar pustules, following impetiginous eczema, such as occurs in blennorrhœa neonatorum, and as a diphtherial skin infection, have likewise been observed. The latter affection was chiefly in very young and in debilitated children.

The clinical picture is characterized by progressive swelling and reddening of the lid, accompanied by swelling of the corresponding

FIG. 18.



Gangrene of the lid in vaccine ophthalmia.

pre-auricular glands. Incision of the tense, cedematous lid discloses a lardaceous, whitish infiltration of the tissue which at first is moist and develops later into dry gangrene.

The *treatment* is the same as in anthrax infection. Inunctions with mercurial and with Credé ointments have also been applied.

7. PHLEGMONOUS INFLAMMATIONS AND ABSCESSSES OF THE LIDS

These affections are usually due to streptococcus infection, and often occur in the course of smallpox. Their course, though similar to

that of the same affections of the crown of the head and the forehead, is far more obstinate than even extensive metastatic abscesses on other parts of the skin. Gepner and others observed grave general infection following in the wake of phlegmonous inflammation of the lid.

Furuncles of the lid, which sometimes appear after smallpox, develop as a circumscribed tumor, movable with the skin, and having a yellow dot on the level of the tumor.

The *treatment* consists in a deep horizontal incision and application of bandages moistened with a tepid boric acid solution. The same treatment is indicated in phlegmonous inflammations and abscesses, as soon as the presence of fluctuation establishes the change from infiltration to abscess. Incision is also indicated, in view of the possible danger to the orbit and meninges by suppuration spreading to the deeper parts.

8. HERPES OF THE LIDS

Herpes vesicles, due to disturbed digestion, and herpes labialis in influenza, may also occur at the lids, but disappear without leaving a trace. On the other hand, radiating scars may be formed in unilateral herpes zoster ophthalmicus (Plate II, Fig. 4), which attacks the lids and their vicinity, and is accompanied by acute inflammation of the corresponding pre-auricular and submaxillary glands. This affection occurs very seldom in children. It is usually observed in the region of the first and second branches of the trigeminus (upper lid, forehead and nose; or lower lid, superior maxilla and zygomatic process). The region of the third branch of the trigeminus is very rarely involved. Inflammations of the cornea develop very often, while the sclera, uvea, and apparently also the optic nerve are less often involved. These inflammations are accompanied by lingering anæsthesia or neuralgia of the trigeminus, sometimes by both, together with a lessening of the normal tension of the globe, elevation of the temperature, sweating of the affected parts of the face, and paralysis of the facial, abducens, or of the motoroculi nerves.

The *treatment* is symptomatic, with due regard to the etiology. A cure will be effected and scar-formation prevented, as far as feasible, by powdering the affected places with rice starch, mixed, if desirable, with equal parts of zinc oxide. The crusts resulting from the drying vesicles and pustules should be covered with a boric zinc paste (acid. bor. 2.0, zinc oxyd. alb., amyl. pulv. āā 2.0, vasel. 6.0, lanol. 9.0). Neuralgia of the trigeminus, which usually precedes the eruption of herpes zoster, is treated with the electric current. Should this prove unsuccessful, internal administration of the salicylic preparations, phenacetin, antipyrine, if necessary, morphine injections, are the indicated remedies.

9. TUBERCULOSIS OF THE LIDS

Tuberculosis luposa (*lupus vulgaris*) occurs most frequently in the lower lid, and is often an extension from adjacent parts of the face. Persistence of the affection causes ectropion secondary to destruction of the superficial and deeper layers of the lids. The resulting corneal ulceration often leads to blindness of the affected eye. Primary lupus, which is comparatively rare, is usually located at the inner canthus and the lower fold of the lid.

Lupus is treated by exposure to the X-rays, Finsen light, and radium; by excision, galvanocautery, thermocautery, and the galvanic current. In addition to these measures, tuberculin has latterly been tried again, with such good results that its use seems thoroughly justified in conjunction with the other methods of treatment. Both treatments are best left to the ophthalmological specialist, especially as in secondary lupus of the lid the removed parts often have to be replaced by a blepharoplastic operation.

Tubercular fistula formation of the skin, due to scrofulotuberculous ostitis and periostitis of the orbital margin, often leads to perforation and ectropion, and is most frequently observed at the external half of the lower orbital margin.

Local Treatment.—Incision of the fistula, removal of the tuberculous granulations, injections of iodoform-glycerine, and tamponage with iodoform gauze.

Tuberculous ulcer of the skin (*tuberculosis ulcerosa miliaris*) is rare. It has occurred with tuberculosis of the lachrymal sac, of the mucous membrane of the nose, and also with scars of disintegrated tuberculous lymph-glands and pulmonary tuberculosis. It starts as a nodule which rapidly disintegrates by ulceration, is usually located in the middle half of the margin of the lower lid, often resembles a hordeolum, and may cause loss of the cilia at the affected spot, and slight swelling of the pre-auricular gland.

The tarsus of the affected lid, or opposite points on the upper and lower palpebral margins, may be involved by direct contact; or the tuberculous ulcer of the palpebral margin may owe its origin to one on the conjunctiva. The soft consistency of the ulcer and of its vicinity, as well as the slight participation of the regional lymph-glands, serves to distinguish the condition from primary ulcerative chancre of the lid.

Local Treatment.—Remove with a sharp spoon and powder with iodoform. Careful touching with carbolated lactic acid applied with a small brush is also to be recommended. (R. acid. carb. 5.0, acid. lact. 15.0, glycer. puriss. 20.0.)

Tuberculosis colliquata (*scrofuloderma*) is an extremely rare affection and, according to v. Michel, occurs exclusively in children. It

consists of a nodule about the size of "a hazel-nut, pigeon's egg, or bean," is movable under the skin, or appears in the shape of a circumscribed, ridge-like eminence at the lower lid or the palpebromalar fold. The skin of the affected part is of a soft, doughy consistency and of a "livid blue to violet" discoloration. The skin is centrally injected and, after perforation and evacuation of more or less liquid pus, an ulcer develops with bluish-red, overhanging margins. Its base is made up of flaccid granulations, which occasionally protrude through the perforated spot. It may also extend toward the tarsus. The scar usually forms very slowly, and has a more or less finely serrated or reticular appearance.

Treatment.—Transverse incision of the growth and removal of the spongy granulations with a sharp spoon. Microscopic examination of the granulations shows giant-cells and tubercle bacilli.

Cold abscess, due to periostitis or ostitis of the orbit and the orbital roof, appears principally in the deeper parts of the lids or of the outer canthus. The treatment consists in puncture, aspiration of the pus, and injection of iodoform emulsion into the puncture. Failing in this, the growth should be incised.

In regard to involvement of the lids in tuberculosis of the conjunctiva, compare page 181.

10. SYPHILITIC AFFECTIONS

The initial induration occurs usually at the inner canthus, this region being most frequently touched and rubbed. It occurs less often at the outer canthus and on the lower lid, the intermarginal fringe, excretory duct of the meibomian glands and the upper lid, and quite exceptionally at opposite points on the two lids. The process may destroy the cilia and spread beyond them to the palpebral conjunctiva, extending less frequently from the canthus to the naso-lachrymal region or plica semilunaris and caruncle. The lymph-glands are painfully swollen and indurated at an early period, especially the pre-auricular, submaxillary, and cervical ones.

Infection of the lids is favored by such loosening of the epidermis as frequently occurs. This may be due to impetigo of the lids, inflammation of the meibomian glands, excoriations and fissures resulting from blepharitis, paralysis of the facial nerve, affections of the secretory apparatus of the lachrymal sac, and affections of the conjunctiva, catarrh, etc.

Children are most frequently infected by kisses from syphilitic individuals, or by accidental lesions from finger-nails, infected gloves, towels, aprons, underclothing, etc. Hard chancre of the lid has occurred from moistening the eyes with saliva, licking them with the tongue to remove foreign bodies or cure some affection. Auto-inoculation is very

Chancre of the lids may appear as a coarse, circumscribed, dark-red papule, or a flat, reddish induration occupying the entire lid. It is usually more painful here than at any other point, owing to its more rapid and intense development, and also because the lid is plentifully supplied with nerves. As a rule, the neighboring palpebral conjunctiva is affected by catarrh, and there is a serous swelling of the scleral conjunctiva. Nodular infiltrates with suppurative disintegration often occur in the vicinity. The induration itself may persist for a long time, sometimes for more than a year.

Owing to the coarsely elastic, hard, or cartilaginous consistency of the base and of the edges of a primary chancre of the lid this, with its subsequent ulcer, cannot be mistaken for folliculitis or perifolliculitis of the marginal glands. Other distinctive marks of the chancre are the high degree of inflammatory swelling of the affected lid and its neighborhood, the short period of incubation (two to four weeks), early and considerable swelling of the lymph-glands, especially the pre-auricular and submaxillary ones, and the manifestation of other syphilitic symptoms. Secondary and tertiary manifestations of the affected and the unaffected eye (uveitis, etc.) may likewise occur. In doubtful cases the result of antisiphilic treatment will decide the question.

Chancre of the lid usually heals without leaving disfiguring scars, but unless it is correctly diagnosed and competently treated at an early period, it may cause deformity of the lid and symblepharon.

Treatment.—Constitutional treatment with mercury, etc. When there is considerable erosion, the local treatment should consist in powdering with iodoform, iodofan, or xeroform; application of ung. cinereum, emplastrum cinereum, or white precipitate ointment. The latter remedies are applied on borated lint in such a way as not to irritate the conjunctiva. The treatment of displaced lids and symblepharon is given on page 106.

As to the eruptive forms, such as macular and papular syphiloids, roseola, etc., the squamopapulous variety appear more frequently at the outer canthus, as desquamative blepharitis—acne or seborrhœa sicca. The palpebral margins, especially the upper ones, are slightly reddened and puffed; the lids, and sometimes the brows, are covered with small, white or grayish scales, like those on the head. In other cases the palpebral margins are covered with yellowish crusts, owing to increased secretion of the sebaceous glands. There is an itching sensation in the eyes, and an increased susceptibility of the eyes to atmospheric changes, especially in overheated or badly-ventilated rooms.

There are similar manifestations, it is true, in ulcerating blepharitis, but that occurs only rarely. Syphilis may cause small scars on the palpebral margin, with a bending inward of the cilia, or the latter may

be destroyed temporarily or permanently. The same process occurs in syphilitic alopecia of the head, or sometimes of the whole body, but the cilia are most frequently involved.

Large papules occur very often at the fold of the upper lid, at the outer or inner commissure, or on other parts of the lids, simultaneously with others which appear at the angles of the mouth and at the external auditory duct. Should these papules ulcerate, they cause atrophy or loss of the cilia, possibly in conjunction with trichiasis and small sinus-like defects of the palpebral margins. Ulcerated papules of the lids and conjunctiva, sometimes accompanied by others on the brows and foreheads, are often the only signs of a syphilitic relapse. If present at opposite parts of the palpebral margins, they will give off an exudation and ulcerate, but owing to the simultaneous presence of catarrhal irritation of the conjunctiva, they easily escape recognition.

It is important in a differential diagnosis to observe that as soon as the disease begins the cilia can be very easily extracted, and that they will be shed, with or without disintegration of the papules. Obstinate catarrh of the conjunctiva may be present and will subside only as the cilia are replaced. The mucous patches at the external commissure of the lid may resemble two papules, owing to the resulting fissures.

Treatment.—Soften the scales and the accumulated secretions with tepid 3 per cent. borated bandages and remove the deposits by means of cotton pledgets saturated with pure olive oil. The margins of the lids are then covered with a thin layer of white prec. salve or white oxide of zinc, or with neutralized plumb. acet. (0.05–0.1, vaseline-lanolin $\bar{a}\bar{a}$ 5.0). These applications should be renewed every two or three weeks, in case their continuous use is indicated. Should there be considerable hyperæmia of the palpebral margins, after removal of the secretion, they should be powdered with equal parts of white oxide of zinc and amylum. Careful touching of the ulcerating places with a finely-pointed stick of argent. nitrico-chlorat. may also have a favorable effect upon the subsequent conjunctival catarrh.

Edema of the lids, as a late manifestation of syphilis, will disappear under specific treatment.

Gummatous syphilides occur either as a superficial, smooth, extensive infiltrate, or as a deep, hard, circumscribed nodule in the subcutaneous connective tissue of the lids. They may resemble a hordeolum. These nodules are sometimes as large as an almond, or even larger. They may occur in multiple form and in both lids. Their growth is either rapid, with febrile manifestations, puffiness and reddening and painfulness of the lid, or they progress slowly, without giving rise to any reactions. The next sequela is an indurated tumor of dirty gray color, with serrated and undermined margins, spreading to the free palpebral

margin and the tarsal conjunctiva, as well as from one canthus to the other, or toward the frontal, nasal, and temporal regions. The affected lid is rigid and hard, and can be everted only with difficulty. There may be retracted, white, radiating scars, with more or less atrophy of the skin or even cartilage formation, leading to loss of the cilia and varying degrees of coloboma of the lid. However, recovery without ulceration has also been observed. Subcutaneous gummata of the lid may cause destruction of the palpebral cartilage and conjunctiva, perforation of the lid, corneal ulcers, symblepharon, or corneoblepharon.

An ulcerating initial induration and an ulcerating gumma are difficult to differentiate, as they often present the same external appearance. In both forms the lids from the margin to the tarsal part are as hard as a board, impairing their function and causing partial or total loss of the cilia (madarosis). The following data, however, will decide the diagnosis: the history, complete physical examination, previous treatment, swelling of the neighboring glands, secondary manifestations of former ocular affections, such as iritis, etc., gummatous growths in other parts of the body, or other late syphilitic symptoms, and the Wassermann reaction. After a gumma, an extensive, deep adhesion will persist; after a chancre, a flat, less extensive scar will remain or even none at all. Gumma of the lid has also been mistaken for carcinoma, hordeolum or chalazion, fibroma, lupus or tuberculosis of the lid, and *vice versa*. The real facts have often been cleared up by other symptoms that came to the rescue. Signs of hereditary syphilis have been observed in infants and young children, such as loss of the cilia, brownish-red discoloration of the palpebral papules, small ulcerating papules of the palpebral margins, and "tuberculous" pustules which change into serpiginous ulcers.

The general treatment consists principally in bathing with sublimate and the use of iodine preparations. The local treatment is the same as outlined for the primary affection (page 83).

An affection of great rarity is gummatous syphilitic tarsitis in hereditary or acquired syphilis. It may occur in the shape of considerable uniform, more or less indurated thickening of the palpebral cartilage, or it may be confined to certain parts of the tarsus. Generally it runs a painless course, and may terminate after a few months, with a remarkable softness and smoothness of the tarsus. One upper and lower lid are often attacked simultaneously, but v. Michel also observed the affection in each lid in succession.

In order to differentiate the affection from hordeolum, chalazion, and amyloid tumors, the following points should be noted: the history, residues of the primary affection, glandular swelling, eruptions of the skin and other signs of syphilis, the favorable influence of etiological

treatment, and the fact that in syphilitic tarsitis the inflammation usually remains localized on the tarsus, spreading but slightly to the lid. Adhesions of the lid, loss of cilia, and eversion of the lid margin do not occur, except when there has been considerable loss of tissue. Nor is the dark-red or bluish tense skin of the lid adherent to the cartilage. The latter generally has a smooth surface, is not painful, and can be distinctly recognized by its half-elliptic shape. The tarsus may be so much infiltrated that the lid cannot be everted. A large portion of the cilia may either be lost, or there may be a second row of fine hairs which take an abnormal direction outward or else turn onto the eye (distichiasis). The pre-auricular and submaxillary lymph-glands of the affected side are usually swollen but do not suppurate. The corresponding part of the cul-de-sac and palpebral conjunctiva is often reddened, finely granulated, or thickened like velvet. Should the inflammation extend to these parts, they assume a pale yellow or yellowish-white appearance. The ocular conjunctiva is normal in most cases or moderately injected. In other cases it is slightly oedematous, and simultaneously the seat of a syphilitic infiltration.

Acute syphilitic tarsitis is a swelling of the cartilage which may be very painful. It occurs with hard chancre of the conjunctiva, and must be regarded as a secondary, inflammatory infiltration directly caused by the latter. Acute tarsitis has also been observed as an early symptom of syphilis, but this is rare.

The *treatment* in the first place is constitutional; this is often aided by local painting of the lids with tincture of iodine, to reduce the tarsal infiltration. As the tincture penetrates rapidly in all directions, it should be applied very carefully. Nor should it be applied near the palpebral conjunctiva. A finely-pointed brush should be used, from which part of the tincture is wiped off. After the painting, borated vaseline is applied on lint.

II. SOFT CHANCRE

A **soft chancre** (*ulcus molle*) may occur at the same place as the hard variety and also be accompanied by inflammation of the neighboring glands. It is caused by auto-inoculation, oftenest by soiled fingers. Powdering the ulcer with iodoform or sozoiodol will usually effect a cure in from two to four weeks. There remains a permanent soft, flat scar and, if the ulcer has spread to the neighboring conjunctiva, an indentation of the palpebral margin at that point.

Cauterizing with silver nitrate has been done in fresh cases; in more advanced cases, Welander recommends hot fomentations (40° C.), the lid being protected by a layer of borated lint moistened with tepid water. Antiphlogistine might do even better.

12. GLANDERS

This disease has been observed on the lids in the shape of pustules, the size varying from a pea to that of a hazel-nut or walnut. The pustule rests on a red, indurated, and painful base. It has also been shown bacteriologically that glanders has been the cause of an ulcer at the inner canthus, which had spread to the ocular conjunctiva, and was followed by a tumor on the cheek with abscess formation, and by purulent ulcerous processes at other parts of the body, accompanied by slight elevation of temperature.

Krajewski observed an ulcer due to glanders on the right lower lid, and an indurated nodule on the left upper lid, with desquamation of the affected tissue. This happened to a child who had come in contact with straw used in a stable where horses with glanders had been quartered.

In dubious cases tuberculous or syphilitic ulcers of the lid may be excluded by bacteriological examination, by serodiagnosis, and by the intraperitoneal inoculation of a guinea-pig with the secretion from the ulcer.

Treatment.—After infection of the lids and conjunctiva with the secretions of an animal suffering with glanders, the lids must be instantly and thoroughly cleansed with sublimate. The conjunctival sac, after being made thoroughly accessible by eversion of the lids, is rinsed with sublimate (1 : 5000). A 2 per cent. silver nitrate solution is then instilled. Dissection of the nodules and ulcers is best left to the ophthalmic surgeon, as is the removal of the lid deformities resulting from later contractions.

13. ACTINOMYCOSIS

Partsch observed this affection in the lower lid of a girl of fifteen. It had extended directly from the upper maxilla, and had also appeared in the upper lid. The latter was swollen in the shape of a single hard nodule the size of a hazel-nut.

The *diagnosis* depends in the first place upon the demonstration of actinomyces and fully-formed actinomycotic cells at the point of infection. This is a difficult matter if the primary focus has healed, or if, for instance, a carious tooth has been removed. Swelling of the lymph-glands is not sufficient proof for a diagnosis.

Treatment.—If the point of infection is distinctly circumscribed, it is removed with knife and scissors, the resulting wound being sutured, if possible. If the edges are not distinctly marked, the granulated tissue is removed with the sharp spoon, followed by a tampon of iodoform gauze. The directions given on page 78 to prevent shortening of the lids in anthrax hold good for the present affection as well.

14. LEPROSY

In this disease flat or nodular, indistinctly marked infiltrates can be felt in the œdematous lid, or small cutaneous lipomata located on the palpebral margin. The lids may be affected first, often symmetrically; in other cases the leprosy spreads from the skin, forehead, or eyebrows. The tarsal conjunctiva and the globe may be infected from the margin of the lid.

The usual consequences are trichiasis, loss of the cilia and eyebrows, softening and disintegration of the nodules, decreased sensation of the skin, and scars. The latter cause atrophy of the orbicularis muscle and consequent paralytic lagophthalmos, often accompanied by obliteration of the puncta lacrimalia, permanent traumatic conjunctivitis and keratitis, or paralytic ectropium and cicatrization of the lids.

To establish the diagnosis, it is necessary to demonstrate the presence of the leprosy bacilli in the hair-follicles or in some other part of the face.

Treatment.—In addition to warm baths, the following remedies have been recommended: Arsenic, quinine, iron, cod-liver oil, sodium salicyl., iodipin, chaulmoogra oil (as an inunction, internally and subcutaneously), antileprol, pyrogallol, benzoynastin, X-rays, and early excision of the nodules, followed by careful cauterization of the traumatic surface by incandescent platinum wires.

Engel prescribes antileprol 2–5 Gm. daily according to age, or in gelatine capsules containing 0.25 to 0.5 and 1 Gm. Young children receive the preparation in drops, which are readily taken in hot milk or in an infusion of fennel or caraway. The treatment has to be continued until a cure, or at least objective improvement, has been effected, but in no case for less than one or two years.

Formora recommended massage with a 10 per cent. airol-vaseline ointment, and injection into the leproma of a glycerine-airol emulsion (airol 5.0, glycerine 35.0, aq. dest. 10).

Cilia which have acquired a faulty position have to be removed to prevent corneal irritation.

The palpebral margins are freshened up and sutured at the inner or outer canthus, as in tarsorrhaphy, in order to prevent lagophthalmos and ectropion.

For safety, careful examinations should be made of the mucous membrane of the nose and of its secretion in apparently healthy children and adults who live with leprous individuals. Obstinate nasal catarrh, commencing ozæna, and severe bleeding of the nose should be suspected as initial symptoms. The removal of the nasal affection is the first and most important task.

15. ECZEMA OF THE LIDS

Children are particularly susceptible to all eczematous affections, and especially to such inflammations involving the lids. In a general way, the causes are diminished power of resistance, weak constitution, anæmia, rhachitis, and scrofula, on the one hand, and overnutrition with gastro-intestinal intoxication, on the other. It is also thought that squamous and ulcerating blepharitis, impetiginous eczema of the lids, and the exuding eruptions of the external canthi, are causative factors because the skin over the lid is not sufficiently resistant to external influences by reason of anomalies of nutrition. Such germs as the *Staphylococcus aureus*, the *Streptothrix*, and *Bacillus refringens* are present in the upper epithelial layers, the pores of the skin, the ostia of the glands and hair-follicles, and at the cilia of the normal lids. They are usually harmless, but sometimes become pathogenic and apparently participate—at least secondarily—in ulcerative blepharitis and other affections. The palpebral margins, notably at the canthi, are exposed also to increased irritation by the “diplobacillus conjunctivitis,” or to trachoma of the conjunctiva, and to eczematous scrofulous conjunctivitis and keratitis. Besides, there may be retention of the lachrymal fluid due to photophobia. Rubbing the eyes with the finger or handkerchief to relieve the itching, which is so often present in eczema of the lid, is also irritating. The secretion of the lachrymal fluid is often impeded by serious catarrhal changes of the lachrymal duct or mucous membrane of the nose (dacryocystic blennorrhœa), and by strictures (polyps), which favor the occurrence of ciliary folliculitis (sycosis), usually of the lower lid. This refers particularly to the unilateral inflammations of the palpebral margins. Increased secretion of the sebaceous glands in non-bacterial chronic inflammation of the palpebral margins—white and dry seborrhœa—will cause the formation of whitish scales which look like dandruff between the lashes (eczema seborrhœica, squamous blepharitis; Plate I, Fig. 5). These may either be dry or saturated with fat. The skin of the eyebrows and scalp is usually similarly affected. In actual acute eczema the margins of the lids are reddened, puffed, and covered with superficial crusts. As desquamation sets in, small vesicles and sycosis-like pustules will appear, each of which seems centrally perforated by one ciliary hair. The eyebrows are often changed in like manner. This condition may now undergo a healing process. If, however, it has been of long standing, or if the canthi are continually moistened by conjunctival secretion, an exudating or squamous eczema will appear at the canthi, and spread to the point on the upper lid corresponding to the fascia tarso-orbitalis (eczema intrageminosum). Scratching and similar irritations will cause furuncular, ulcerative loss of sub-

stance, which, after the closely adherent crusts have been removed, leaves a serrated palpebral margin (secondary infection; Plate I, Fig. 4). Here, again, the lighter cases may take a favorable course, while in serious and deep ulcerations there may be a phlegmonous erysipelatous inflammation, often leading to general atrophy and loss or blanching of the cilia, or trichiasis. There may also be a persisting œdema of the lids and pachydermic infiltration of the skin, notably of the upper lid, which refuses to yield to treatment. This is often due to an acute eczema of an erysipelatous character which may attack the whole of one or both lids, of one or both eyes. The entire neighboring skin of the face may be involved, and the pre-auricular glands swollen. If the course is relapsing and chronic, the result may be a chronic puffiness of the lids, notably the upper one, causing it to droop and in some degree cover the lid fissures.

The *prognosis* in all these affections, which often have a tendency to relapse, depends upon the general treatment, upon the removal of any possible cutaneous affections of the head and face, such as eczema, pediculosis, or favus, and upon the local measures instituted. This refers particularly to the ulcerative eczemata of the palpebral margins, whose cicatrization may cause obliteration of the intermarginal space or a slight cicatricial ectropion of the lower lid, atrophy, and malposition of the cilia. The chronicity of the affection is favored also by the frequent presence of phlyctenular and pustular processes on the conjunctiva and cornea, just as the sequelæ of eczematous blepharitis favor madarosis, trichiasis, tylosis, and ectropion.

Treatment.—In the first place, any eczema of the eyebrows, scalp, ears, and lips must be thoroughly treated. This applies especially to fissures on the upper lip and nostrils, which often accompany or precede eczema of the lids and keep it alive. Touching with *argentum nitro-chloratum* effects a quicker cure than ointments. It is important, however, to see that the cauterized places are not mechanically irritated afterwards, as by blowing of the nose, and that there are no cracks beneath the surface. Covering these spots with a thin layer of salve will favor desquamation. Removal of the swelling of the lymph-glands and of any tuberculous manifestations of the joints or bones is of equal importance. The persistence of the affection is due to want of cleanliness, improper diet, and living in hot or badly-ventilated rooms. A change in this respect may suffice gradually to ameliorate the condition as years go on. Pathological changes in the lachrymal sac, nose, and nasopharyngeal space should be treated (compare page 121). The lid margins should be kept scrupulously clean, using a mild neutral soap, such as the Hebra soap. Cold bandages are contra-indicated, as they aggravate the palpebral eczema.

As to the special treatment of these various conditions, the following rules should be observed: in chronic, so-called dry eczema, with more or less pronounced desquamation, paint the margin of the lid with ol. cadini, ol. fagi, or ol. rusci (1 : 10 ol. olivar.), taking care that none gets into the palpebral fissure. Or, apply a 5 per cent. ichthyol ointment, or a paste made of ol. rusci 0.5 and Lassar's paste 20, as follows: place a little ointment, no larger than a small pea, on a disinfected glass rod, and apply it to the lid, distributing it so that it will just reach the anterior border of the intermarginal edge. Any superfluous ointment is removed with a blunt carrier of sterile hydrophile gauze.

In wet eczema (ulcerating blepharitis), cleanse the skin of the lids thoroughly from scales and crusts, both morning and night. Soften it with a moist boric water bandage, which is allowed to remain for five or ten minutes; then gently and slowly wipe off the lids with sterilized cotton pledgets, saturated with olive oil. To grease the palpebral margins, apply in the morning a cooling ointment, such as lanolin. anhydric. puriss. "Liebreich" 5.0; aq. plumb. duplex (free of carbonate) 5.0. This may be used with hydrochloric cocaine 0.05, freshly prepared for each case and kept in a porcelain jar with a closely-fitting celluloid cover. If the skin adjacent to the temporal palpebral commissure tends to crack, grease it slightly and separate it with the thumb and index-finger of the disengaged hand. In this way every part of the lids and their surroundings is uniformly softened. In the evening, apply either zinc ointment (zinc. oxyd. alb., amyl. āā 5.0; vaselin. 5.0) or unguent. præc. alb. pultiforme (Hg præc. alb. pultif. 0.1; vassel. 5.0). Von Michel applies a modification of Lassar's paste. In acquired or even in congenital lessening of the height of the lids, apply ointment or paste on lint over the closed eyes, and secure it by a light bandage.

Schlen and Peters advise inunction of the lids in the evening with ichthyol-zinc ointment, and covering them with zinc-ichthyol gauze (ammon. sulfo-ichthyol. 0.2-0.5; amyl. tritic., flor. zinc āā 10.0; vassel. americ. 25.0; M. F. ungt. exact. terend.). Lassar's red mercury sulphide paste (ungt. rubr. sulfurat.) is very efficacious in impetiginous eczema: it is spread on lint with a glass rod, in a layer as thick as the back of a knife. It is placed upon the cleansed lids and kept in position by a bandage.

It often happens, however, that even unirritative ointments are badly borne. The marginal ulcers should then be carefully touched with a finely-pointed stick of argent. nitrico-chlorat. (argent. nitricum, to which 5 per cent. argent. chlorat. is added), and smoothed by wiping with cotton moistened in hot water. This has a more rapid effect than argent. nitric. mitigat. The base of the small abscesses or ulcers has first to be cleansed, and any loose cilia must be gently removed with the ciliary forceps (Plate XI, Fig. 8). This is best done as follows:

Grasp each hair with the forceps, slowly loosen it with corkscrew movements, and extract. If the margin is inflamed, a repetition of the epilation may be necessary. The cilia may be so minute as to require a magnifying glass to detect them, or so fragile as to require a firm hold to extract them. If these methods should prove unsuccessful, after a sufficiently long trial, or if the margin is chronically inflamed and puffed (tylosis), all the cilia will have to be removed. As this is a painful process, anæsthesia may be necessary. The operation may have to be repeated at intervals, until the margin is normal. In acute sycomatous eczema of the palpebral margin, and in abscesses of the hair-follicles, puncture of the pustules and epilation of the cilia, a 3 per cent. boric ointment or Lassar's paste and ointments of sulf. precip. resorcin, or acid. tann. sulf. precip., vassel. amer. alb. pur. 1 : 2 : 20, may be tried. If it is not efficacious, the ulcers should be touched with a finely-pointed stick of argent. nitrico-chlorat.

As to treatment of the affected lachrymal ducts and simultaneous or subsequent irritation of the conjunctiva, see pages 121 and 139.

The same treatment, particularly the epilation of the affected cilia, is instituted in syçosis hyphogenes or parasitaria (trichophytia profunda), which is acquired by contact with horses or horse-infected children.

I think it superfluous to extract all the cilia in order to sterilize the skin of the lids in operations on the globe (iridectomy, etc.). Of course, all solutions, such as cocaine, suprarenin, atropin, physostigmine, etc., should be of standard quality, and all instruments used in operations on the eye should be of the best material, including even those for the removal of cilia or foreign bodies from the conjunctiva. The lids and vicinity are best cleansed by thoroughly rubbing the skin with ether or benzine applicators. To cleanse the conjunctival sac the patient is placed on his back and the sac is gently irrigated with oxycyanate of mercury (1 : 2000), or sublammin (1 : 2000), or sublimate (1 : 500). The lids being held apart by two blunt retractors, the patient is instructed to move the eye in all directions during the irrigation, so that all the folds of the conjunctiva may be exposed. Firmly adherent, desquamated epithelium or skin is removed with sterile cotton pledgets. This should precede the instillation of the local anæsthetic, in order to prevent injury to the cornea. Drying of the corneal epithelium following cocaine instillations in the open eye may be prevented by having the eyes closed until the beginning of the operation.

If eczematous blepharitis is associated with excoriations of the external canthus or with palpebral spasms, painting with a 1 to 2 per cent. solution of silver nitrate may be useful. The patient's head is placed in a horizontal position, and a brush of medium size, carefully cleansed, is applied several times with more or less pressure, while the thumb and

index-finger of the disengaged hand separate the lids just wide enough to reveal the fissure. Or, if preferred, a cotton pledget saturated with a freshly-prepared 1 per cent. solution of argent. nitricum may be rubbed horizontally across the lids. This lid massage promotes healing of the excoriations at the external canthus and at the same time benefits the spasms. It should be repeated at gradually-increasing intervals until the hyperæmia of the lid has disappeared. Careful painting of swollen lids with tincture of iodine will often have a favorable effect.

An eczematous inflammation of the lids has a great tendency to recur, and for this reason may require treatment in an institution. This applies particularly to cases that have been neglected, to those complicated by conjunctival or corneal affections, to patients whose nursing and food at home are inadequate, or whose general condition has been much impaired by infectious diseases.

The local treatment of seborrhœa of the palpebral margins is tedious. It is often hereditary, persisting then from childhood to old age, and may be accompanied by seborrhœa of the scalp or other hairy parts of the body. The cilia may be partly or totally lost, leading to irritation and anomalies of the conjunctiva which defy treatment, and may endanger sight by subsequent affections of the cornea. Gentle cleansing of the lid with Hebra soap and tepid boric and borax water in the morning, and the application of carefully-prepared mercurial sulphur ointment or hydrarg. præc. alb. 0.05, vasel.-lanol. āā 5.0, in the evening, should be regularly continued. Von Michel prescribed thin layers of paste consisting of lac. sulf. 1.0, or resorcin. albiss. 1.0 with zinc. oxyd. amyl. pur. āā 10.0, vasel. amer. 20.0, in seborrhœa sicca, to be applied to the palpebral margins with a glass rod scoop. In old cases Haab used tincture of iodine.

Pustulous eczema of the lids is differentiated from impetigo contagiosa by diffuse hyperæmia of the lids in the latter affection. The eczema may occur as a family affection, or endemically in schools and kindergartens. When primary and acute, the lids are surrounded by an inflammatory areola and impetiginous vesicles resembling dew-drops, which dry up into honey-colored crusts. Owing to repeated plucking, the latter assume a yellowish-green or brown discoloration and become confluent. Healing occurs spontaneously in a few weeks, and treatment is therefore merely palliative, such, for example, as $\frac{1}{2}$ -1 per cent. precip. ointment, followed later by Lassar's paste.

16. PHTHIRIASIS PALPEBRALIS

This affection should not be mistaken for eczema of the lids. Its favorite location in childhood is at the cilia, owing to absence of hair of the axilla, pubes, etc. Phthiriasis pubis also nestles in the eyebrows;

...the eyelashes are attached to the cilia, giving them an appearance of being covered with small spots of dust. ...when meeting the cilia, are smaller than on other ...near detection may require the use of a magnifying glass. They cause considerable itching and inflammation, or ...the palpebral margins. They are removed with ...will kill the nits; mercurial ointment, white or yellow ...will kill the lice. Observance of general cleanliness ...occurrence.

17. HORDEOLUM (PLATE I, FIG. 6)

Suppurative inflammation of the glands of the palpebral margin is called hordeolum externum; that of the meibomian glands, hordeolum internum. The latter occurs mainly in the tarsal tissue; it does not yield readily to treatment, and is therefore much more painful and tender than the former. The neighboring skin of the lid and the cutis and ocular conjunctiva is more or less œdematous and hyperæmic, resembling erysipelas. Should there be considerable conjunctival secretion, the less experienced physician may be deceived into mistaking the affection for incipient ophthalmoblennorrhœa, especially if the neighboring bulbar conjunctiva is chemotic.

If several sties exist, the lids may be so swollen as to interfere with opening of the eye. The initial affection is differentiated from acute inflammation of the conjunctiva by pain and tenderness at a more or less circumscribed spot.

The pus perforates the conjunctiva rather than toward the skin of the lids.

Eczema of the skin of the palpebral margins, chronic catarrhal conjunctivitis, affections of the lachrymal ducts, and continued wearing of a binocular bandage predispose to staphylococcus infection of the palpebral margin or its vicinity. It may also simultaneously affect other parts of the face. This occurs chiefly at puberty coincident with anaemia and intestinal auto-intoxication (acne juvenilis). If the nodule is deep seated, the pre-auricular gland of the same side may be swollen.

Treatment is directed to the removal of the cause. Yeast preparations, such as cerulin, levurinos, levuretin, zymin, or fresh beer yeast and flores. sulf. sacchar. lact. $\bar{a}\bar{a}$, are administered internally. Local treatment must deal with the affected conjunctiva and lachrymal sac. The new pustules are evacuated by a superficial incision, followed by painting of the skin with borated vaseline.

Acne varioliformis (necrotic type), a very rare affection, is generally unilateral and affects the cutis of the lids. In one case it has been observed simultaneously on all the four lids. It also attacks the region

of the brows and the cheeks. After desquamation of the dried pustules, circular, sharply-defined, slightly-indented scars will remain.

The *treatment* consists in softening the crusts with oil. After their removal, the affected places are covered with sublimate vaseline (1 : 5000) spread on lint.

In folliculitis ciliaris of the infectious necrotic type there are itching of the lid, fever, general debility, and swelling of the cervical and pre-auricular glands. On the following day pustules appear over the entire palpebral margin, which by this time has become uniformly indurated. The pustules will burst, taking the cilia away, and healing several weeks later with scar formation.

In hordeolum internum, softening of the nodule by local application of heat is a useful start. Yellowish discoloration of the palpebral conjunctiva indicates an imminent eruption of the pus inward. The affected spot is then incised in a vertical direction in the conjunctiva tarsi, which is exposed by everting the lid. Care must be taken not to injure the tarsus or the acinous sebaceous glands embedded in it, as they are important for lubricating the palpebral margins. Borovasoline ointment, three per cent., the size of a pea, is applied twice or three times daily to the conjunctival sac. Instructions should be given not to touch the lids. To prevent recurrence, frequent cleansing of the lids and eyebrows with soap and internal application of the remedies mentioned on p. 94, especially yeast preparations, are of importance. The treatment of blepharitis should be persistent if the object is to effect a thorough cure. Haab advises the instillation of zinc drops into the conjunctival sac once daily after the inflammation has subsided. By the addition of sublimate (zinc. sulf. 0.025 : 10; solut. sublim. puriss 1.0 : 5000 or 1.0 : 10000 āā), this will act as a disinfectant also.

18. CHALAZION (PLATE II, FIG. 2)

A chalazion is the result of an acute or chronic affection of the meibomian glands, caused, according to the investigations of v. Michel and Waetzold, by the toxic effect of bacteria that have been destroyed by phagocytosis. It is associated with other ocular affections, such as conjunctival catarrh, stenosis of the lachrymal ducts, hypermetropia, and astigmatism; also with constitutional weakness, such as the tuberculous and scrofulous diathesis. It is rare in childhood. Unlike an hereditary syphilitic, gummatous, or tuberculous infiltration of the tarsus, a chalazion usually develops painlessly and slowly. The swelling is circular or transversely oval like a circumscribed nodule, over which the skin is more or less movable. It is elastic, while a gummatous nodule is cartilaginous and hard. In a tuberculous nodule, swelling of the lymph-glands and a similar affection of the conjunctiva are rarely absent.

Sometimes, however, it requires careful general examination or an exploratory incision to arrive at a definite diagnosis. It often happens that a chalazion shines more or less extensively through the tarsal conjunctiva, displaying a grayish-yellow discoloration. In other cases the tarsal conjunctiva is red and swollen in the neighborhood of the transparent place. The chalazion may rupture into the tarsal conjunctiva, or at the palpebral margin, into an excretory duct of a meibomian gland (chalazion marginale).

Treatment.—After the application of cocaine the lid is everted so as to expose the point of the tarsal conjunctiva corresponding to the chalazion. This place is touched with a very fine, slightly incandescent galvanic point until the mucus or mucopurulent granulation tissue of the cystoid growth is evacuated. The remaining masses and the resistant capsule are destroyed by inserting the incandescent needles several times, more and more deeply, at the same time slightly enlarging the opening of the tarsal conjunctiva. The after-treatment is with heat, cataplasms, etc. Any small granulations which appear later at the place of puncture are carefully touched, after eversion of the lid, with a finely-pointed stick of argent. chlornitrico., or with a fine conical sound, such as is used for dilating the lachrymal ducts, fused with argent. chlornitrico. The crusts from the cauterization are neutralized by a one per cent. salt solution, but in most cases it is sufficient to keep the lid away from the eye for half a minute or a minute.

A chalazion which inclines to rupture through the palpebral skin, usually near the intermarginal fringe and the ciliary floor, can be opened from without by galvanocautery and removed almost painlessly, after spraying with ethyl chloride (comp. p. 91). The resulting scars can hardly be recognized. I have often applied this method, and find it quite as simple as an incision. The latter method is carried out by incising the chalazion from without by a vertical cut which is afterward sewn up. The incision may also be made from the tarsal conjunctiva in a vertical direction, parallel to the longitudinal axis of the meibomian glands. This is followed by scraping with a sharp spoon, to prevent recurrence. The after-treatment is the same as that already described. Contraction of a chalazion, when opened by galvanocautery, may be accelerated by massage of the lids.

Elschnig states that a chronic conjunctivitis often causes an acute affection of the meibomian glands. In that case the pustules at the mouths of the glands contain the same kind of pus or sebaceous threads as are also found in the conjunctiva. Further investigations concerning this point are desirable. The glands as well as the tarsus may also be secondarily affected by blepharoconjunctivitis or by diphtheria of the conjunctiva.

A chronic suppurative inflammation of the meibomian glands, extending more to the skin of the lids than toward the conjunctiva, was observed by Natanson. It was accompanied by multiple abscesses which had no tendency to perforate spontaneously. Castelain observed multiple infection of the glands with some form of streptothrix, which at first resembled actinomycosis.

19. FAULTY POSITION OF THE CILIA (TRICHIASIS AND DISTICHIASIS)

Permanent faulty position of the cilia may be due to scars or inversion of the margin of the lids. This may be the result of ulcerative blepharitis, trachoma, hordeolum, conjunctival diphtheria, or it may come from burns or operations on the lids or conjunctiva. These defects are removed by marginoplastic and operative treatment of the entropion, primarily to prevent or remove any irritation of the conjunctiva. They may lead to abrasion of the corneal surface.

In partial trichiasis, if the cilia are removed, they will grow sooner or later, and therefore they should be destroyed by electrolysis, together with their follicles. The anode, consisting of a cushioned plate, is applied to the temple; the needle, or cathode, is inserted into the follicle. The current is closed from a quarter to half a minute, until a fine froth appears at the ciliary root. This will cause the cilia to drop out, or they may be extracted, if necessary. This being rather a painful process, it is advisable, in sensitive patients, to use local anæsthesia, such as the subcutaneous injection of alypin, or to apply a suprarenal solution, eusemin (cocaine adrenalin), or to spray a little ethyl chloride near the point, but at a safe distance from the operative field. The latter procedure can be carried out only after the lids have been very thoroughly closed.

Acquired alopecia of the cilia has been observed as part of general alopecia following scarlet fever and also in artificial alopecia in an hysterical girl. Unilateral gray or white discoloration of the cilia has been observed after iridocyclitis, or without any definite cause. Ponti produced a new crop of normally pigmented cilia by epilation of the discolored ones, and application of a slightly irritating ointment.

The following are some rarer affections, in whose course the lids were injured in infancy and childhood: Acute pemphigus of the new-born, Darrier's disease, which has so far proved intractable; acute pemphigus such as occurs at the margins and angles of the lids; the malignant, fatal hemorrhagic pemphigus, which is accompanied by an eruption of vesicles on the conjunctiva; the benign simple chronic pemphigus (*pruriginosus foliaceus*), which may be confined to the skin of the upper and lower lids and their immediate vicinity, or may be associated with pemphigus of the mucous membranes and, particularly, of the conjunctiva.

Treatment.—Application of rice powder, boric ointment, or byrolin to the affected skin of the lids; also general internal treatment, especially with arsenic.

Similar treatment is indicated in urticaria caused by auto-intoxication, or when accompanied by considerable œdema of the lids, especially the upper ones.

Hyperkeratosis universalis (ichthyosis congenita), an affection of debilitated, moribund infants, often involves the skin of the lids to a very considerable extent. This affection, as well as ichthyosis simplex, causes tension and shortening of the skin, interfering with closure of the lids, and thereby favoring ectropion of the lower lids, and secondary inflammation of the conjunctiva and cornea. The latter may also be caused by a thin and dry condition of the cilia and eyebrows, with consequent narrowing of the lid fissure or adhesions at the external canthus.

Treatment.—Kaulich recommends soda baths and frequent incunction with borated lanolin. Operative interference in this form of blepharophimosis is not advisable, since canthoplasty, which would otherwise be effective, might favor the formation of ectropion, by additional shortening of the lids.

Xeroderma pigmentosum, which is often observed as a family affection, is as yet etiologically and anatomically unexplained. It may cause ectropion by cicatrization, excrescences, and even malignant ulcers with fatal issue.

In the way of treatment, it has been suggested that patients remain in rooms with red illumination, or wear red or yellow veils, since the affection often begins with diffuse or spotted erythema at parts of the body which have been exposed to the sun or diffused light. The excrescences should be removed as soon as possible.

Scleroderma attacks the skin of the lids or one side of the face. Occasionally it is associated with Basedow's disease. Thyroid and salicylic preparations, subcutaneous injections of thiosinamine, application of 2 per cent. thiosinamine plaster, salicylic ointment, massage, and electricity have been used for its treatment. Thiosinamine, however, is not free from objections in active or recently-healed conjunctivitis and keratitis, since it also starts or rekindles these affections.

Neurotic facial atrophy is usually an affection of childhood. In this, the changes of the skin just mentioned, such as spontaneous discoloration and loss of the cilia, and other ocular disturbances have been observed. Ectropion of the lower lid, depending on atrophic shortening of the skin, constriction of the lid fissure, whitish spots on the upper lids, abnormal pigmentation of the skin, and absence of the eyebrows, have likewise been recorded.

Treatment.—The treatment is palliative, although the direct electric current and paraffin injections in the facial skin have been tried.

20. BLEPHAROSPASM

Spasm of the orbicularis muscle may be a local manifestation or only a part of a general spasm of all the muscles supplied by the facial nerve. It may occur in two forms. One is simply a clonic exaggeration of normal twitching, causing the lids only to be spastically contracted (spasmus nictans). The other is a periodical or permanent tonic contraction and closure of the lid fissure (blepharospasm). Both forms may alternate, or change from one into the other. These spasms may be voluntary, involuntary, or reflex manifestations. The causative factors are many. Among them may be mentioned: functional motor neurosis, organic cerebrospinal affections, cortical epilepsy, hysteria, neurasthenia, traumatic neurosis, migraine, chorea, tetanus, tetany, gastro-intestinal affections, intestinal parasites, or scars in the peripheral course of the facial nerve.

A fifteen-year-old boy who for seven years had been suffering from twitching of the lids and tic douloureux would stutter when the tic was relieved; when the pains were present, he would not stutter. Meynier observed occasional rapid closure of the lids with also a rapid horizontal nystagmus in children from five to twelve years of age who suffered from infectious myoclonia.

Habitual twitching of the lids is most frequent in infancy and childhood, and is attributable to imitation, overexertion, masturbation, anæmia, and hereditary neuropathic taint.

Blepharospasm may be caused by all kinds of irritation of the trigeminus, such as injuries to the face and scalp, carious teeth, chronic catarrhs, polypous growths in the nose, hypertrophy of the tonsils, empyema of the superior maxillary cavity, forcible syringing of the ear, and migraine. Moderate pressure upon the points where the supra- and infra-orbital nerves emerge, or at certain other so-called pressure points of the trigeminus, will relieve or remove the spasm. This spasm may also result from irritation in some remote part of the body, or may even be produced by suggestion. It is often intermittent and usually unilateral, or more pronounced on one side.

Reflex spasms, due to retinal irritation, such as intense light or snow-blindness, are usually associated with a neuropathic tendency, opacities of the refractive media, or myopic affections of the macula.

The *prognosis* and *treatment* of non-inflammatory blepharospasm are definite, in proportion to the possibility of removing the cause, either by constitutional or local treatment. Removal of pathogenic factors which are irritating the trigeminus is just as important as the

prescription of suitable glasses. The most usual internal palliative remedies are the bromides, arsenic and narcotics, morphine injections, and chloroform inhalations. Resection or avulsion of the supra- or infra-orbital nerve, removal of the gasserian ganglion, peripheral stretching of the facial nerve, alcohol injections into the facial nerve at the stylomastoid foramen or into the basal part of the trigeminus, are rather heroic measures, which should be advised with caution; they sometimes produce good results, but they have also led to extensive degeneration of the nerves, and to serious paralysis of the facial nerve, with complete degeneration.

Oppenheim uses the galvanic current in four different ways. These are (1) to place an anode which is about 10 centimetres square over the nerve-trunk, with the cathode preferably on the nape of the neck, and

FIG. 19.



Blepharospasm, with acrofulous eczema.

then slowly close and open a weak current of about two to three milliamperes; (2) to place the anode over the occiput, and the cathode at some distant point; (3) both electrodes over the mastoid processes; or (4) the anode over the various branches of the pes anserinus major. If any points of tenderness exist, the anode is applied to them.

Cure of the so-called essential and nearly always hysterical blepharospasm by means of weak faradic, galvanic, and static electricity and d'Arsonval's currents is simply something accomplished by suggestion. The disease is often accompanied by hysterical disturbances of sensation in the region of the orbicularis, such as anæsthesia or hyperæsthesia with pho-

tophobia and lachrymation, and by hysterical ptosis, hysterical restriction of the field of vision, and amblyopia. Massage of the lid in hysterical blepharospasm also partakes of suggestion. The sensation of so-called muscular waves, usually on one side, but of quite a distinct type, does not demand treatment, even if that persists.

Reflex spasm of the orbicularis occurs in irritation or injury of the corneal surface by foreign bodies. In iridocyclitis there may be also sympathetic irritation of the healthy eye. Inflammatory blepharospasm is a frequent and obstinate manifestation of superficial eczematous inflammations of the cornea with epithelial desquamation (Fig. 19). The spasm is usually bilateral, even if the cornea of only one eye is affected. Spasm of the lids, with increased lachrymation and photophobia, may also accompany a recurrence of corneal eczema for weeks or months, and even persist until the corneal affection is cured. Bleph-

arospasm, especially in young children, may cause temporary loss of vision by disuse. Weaksightedness from non-use of the eyes disappears with reacquirement of the lost function in the course of several weeks, after the persistent closure of the lids has been relieved.

Inflammatory blepharospasm is often relieved and gradually removed by continued application of a two to five or even a ten per cent. cocaine vaseline three times daily; or by the instillation of a two to four per cent. cocaine suprarenin, or by immersing the face in cold water three to six times daily, and living in uniformly lighted rooms. The habit which children have of burying their faces

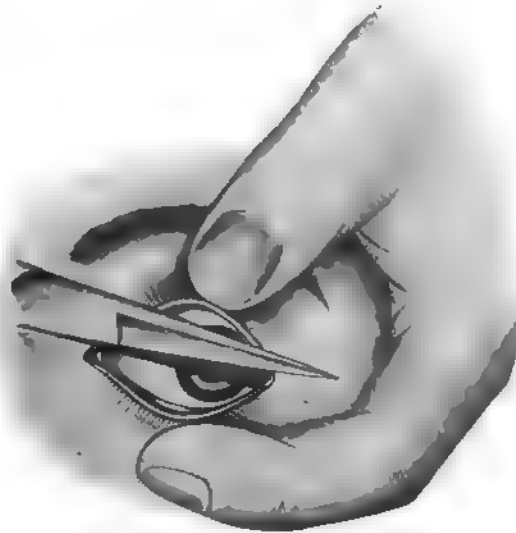
in the pillows, or of lying on the floor with their faces downward, should not be allowed.

Should continued local and general treatment of the cornea and uvea fail to effect improvement, operative interference under anæsthesia is indicated. This consists in enlarging the outer canthus. This is especially advisable when an apparent shortening of the lid with fissures or cracks has led to still greater spasm.

An incision is made with scissors, straight lancet, or bistoury, extending the outer canthus by 10 to 15 mm. toward the orbital margin (Fig. 20); the neighboring parts of the bulbar conjunctiva are loosened, and the lid fissure united by three, five, or seven sutures (Fig. 21). A bandage is applied over both eyes until the sutures are

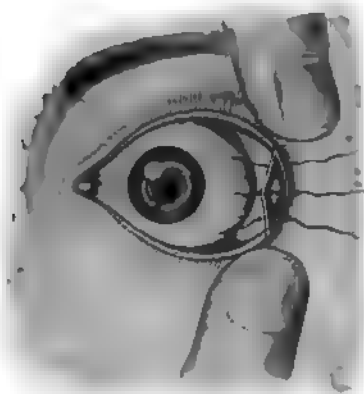
removed. This slight operation requires no special surgical experience. In order to ensure good results, I apply double or treble sutures without

FIG. 20.



Incision of the outer commisure of the lid.

FIG. 21.



Position of the sutures in canthoplasty.

making them fast. They can then be easily removed with small forceps without opening the wound again.

Canthoplasty, as this operation is called, is especially indicated for blepharophimosis or agglutination of the external palpebral commissure,—that is, when cicatrizing cracks have led to gradual constriction of the lid opening. Otherwise there may be stagnation and decomposition of the pathologically changed secretion of the conjunctiva, causing irritation of the conjunctiva, epithelial lesions of the cornea, etc. Blepharospasm owes its existence to such causes. Besides, the maceration of the epithelium prevents healing of the phlyctenular infiltrates and ulcers of the cornea.

FIG. 22.



Normal palpebral fissure.

If this operation is done properly, it will also serve as an excellent means of guarding against the recurrence of scrofulous ophthalmia.

If there is a high degree of blepharophimosis, careful and gentle detachment of the temporal part of the conjunctiva is of the greatest importance. It should be made freely movable in the entire temporal region, and care taken to avoid any pressure on it or puncture of it. It is therefore drawn slightly forward by means of fine forceps. The small scissors, which are bent toward the surface, are first directed straight backward; while undermining the conjunctiva with short strokes, they gradually assume a frontal direction. Bleeding of the subconjunctival vessels will aggravate the detachment, but this may be arrested either at once or after the suture of the commissure has been closed. In suturing, the corresponding places of skin and mucous membrane should be closely approximated, so as to ensure a smooth appearance externally. Folds or ridges are avoided by placing the first suture at the commissure. Then the second and third sutures are placed above and below precisely

in the middle between this first suture and the end of the wound. Following the same plan, any further sutures which may be necessary are placed midway between two neighboring sutures as the case requires.

A thick layer of freshly-prepared yellow ointment is spread upon sterilized, borated lint and applied so as to protect the freshly-agglutinated wound from tearing when changing bandages. The eyes are covered with a thin layer of gauze which has been made pliable with vaseline. Over this layer are placed small pieces of absorbent bandage held in place by a few strips of adhesive plaster. The whole is covered with a binocular bandage. The sutures are not removed before the fifth or sixth day, to prevent a reopening of the wound.

FIG. 23.



Lid fissure after canthoplasty.

A comparison of Figs. 22 and 23 will show how the palpebral fissure, especially the region of the external canthus, is affected by canthoplasty.

21. LAGOPHTHALMOS

Defective closure of the lids may be due to contraction, or to a rough and dry condition of the lids. It may also come from ocular paralysis (*lagophthalmus paralyticus*), either partial or complete. Lid movements are restricted, and the opening cannot be closed voluntarily. Insufficient secretion of the lachrymal fluid may also occur as a complication, as a concomitant manifestation, or facial paralysis with corneal ulcers, etc. Or the affected eye may be completely hidden by the drooping upper lid, due to relaxation of the levator muscle and its own weight. This may also occur during sleep. On the other hand, antagonistic contraction of the levator palpebræ may affect the opening of the fissure, with accompanying participation of the upper lid in the act of raising or lowering of the eye.

If the paralysis of the orbicularis is of long standing, it leads to relaxation of the lower lid (ectropium paralyticum), and to catarrhal conjunctivitis and xerosis of the cornea. The occurrence of these sequelæ is favored by simultaneous paralysis of the trigeminus. Corneal ulcers and loss of epithelium often do not develop because of the fact that the normal direction of the eye is upward and outward during sleep (Bell's phenomenon).

Treatment.—Protection of the eye is most important to prevent necrosis of the cornea. This is effected by wearing spectacles during the day so shaped as to close the lids. At night, if necessary, a 3 per cent. borated vaseline ointment is applied to the lids, the eye is covered with absorbent gauze, saturated with 3 per cent. boric acid, and then with a turn of rubber bandage or with the Billroth's dressing.

The treatment of the many forms of facial paralysis depends in each case upon the etiology. This may mean mercurials, sudorifics, etc., or even operation. After injuries galvanism will sometimes restore the function of the paralyzed nerve. For that purpose the cathode is applied to the nerve-trunk and the anode to the nape of the neck. A current of from 2 to 3 milliampères is slowly closed and opened for two or three minutes. At first this is applied daily, and later every two days. In a long-standing partial paralysis an injection may be made of strychn. nitric. (0.003 to 0.005) underneath the skin in the temporal region. This may be repeated for two or three days, or longer, if required. To prevent undue reaction, the patient should rest in bed for a day or two after the injection. But if muscular contraction should develop, the toxic effect must be neutralized without delay by inhalation of small quantities of chloroform. To prevent as far as possible ectropion of the lower lid, the tears which accumulate in the conjunctival sac should be frequently removed, by passing a clean cotton pledget from below upward, massaging at the same time in the same direction. We may also use occasional instillations of adrenalin, alypin, and holocaine (adren. 1:1000, 1 Gm.; alyp., 0.2 Gm.; holocaine, 0.01 Gm.; aq. dest., 15 Gm.). If this is applied once or twice daily, it often serves to decrease the lachrymation considerably.

The following operation has been successfully employed to reduce the excessive flow of tears: An incision is made into the lower canaliculus, and a V-shaped flap of the mucous membrane is removed from the inner lip of the wound. This is closed with a suture which passes through the caruncle, thus raising the lower lid. In incurable paralysis of the orbicularis it may be necessary to lift the lower lid and shorten the lid opening by lateral or median blepharorrhaphy. This helps to prevent opacities, ulceration or even complete necrosis of the cornea. In pronounced exophthalmos there is a considerable tendency to degeneration of that kind.

22. BLEPHAROPHIMOSIS

Horizontal shortening of the lid fissure, called blepharophimosis, may be simulated by a vertical fold of skin hanging over the outer canthus. It is usually due to slight abrasions of the skin, such as occur frequently in eczematous affections of the cornea, conjunctiva or lids, accompanied by increased lachrymal secretion. As the ocular affection abates, this fold may disappear, provided the skin of the lid reacquires its normal elasticity.

Actual horizontal shortening of the lid fissure (ankyloblepharon acquis.) is caused by adhesions between opposite points of the palpebral borders.

Treatment.—The attempt to separate the lids and to prevent adhesions by applying some bland ointment between the lid margins is nearly always a failure. The only remedy is a canthoplasty after complete cicatrization has taken place. This is especially true if an entropion has also developed. The following methods are especially applicable in typical cases: Canthoplasty with a cutaneous flap (Kuhnt), or horizontal blepharotomy, that is to loosen all the skin of both lids and make a new commissure by uniting the cutaneous flaps (Agnew); or a transplantation of the cutaneous flap may be done according to the Thiersch method; or a strip of mucous membrane may be transplanted from the inner surface of the lips. This last operation especially requires the experience of a specialist. Indeed, all such adhesions of the lid borders as result from lupus, diphtheria, erosions, burns, or other scars of trachoma, or pemphigus, are nearly always associated with some cicatricial ectropion. The plastic replacement of the conjunctiva presents technical difficulties, especially if the conjunctiva is considerably involved, as in symblepharon or corneoblepharon.

23. ENTROPION

In childhood an inversion of the lid border and the adjacent part of the palpebral surface is usually caused by contraction of the conjunctiva. Sometimes, also, a contraction of the whole tarsus may follow deep burns, pemphigus, or chronic trachoma. These contractions cause the ends of the lashes or even the skin of the entire lid to touch the orbital surface. This cicatricial entropion is often accompanied by blepharophimosis, distichiasis, and trichiasis. When it is due to pemphigus, it usually affects the lower lid, while in trachoma either lid is affected with equal frequency.

The *treatment* is always operative. Owing to the possible sequelæ of conjunctival inflammation with loss of epithelium, or ulcers of the cornea, such treatment belongs to the domain of the ophthalmologist.

24. ECTROPION

Eversion of the edge of the lids and of the skin, such as occurs in eczema and in ichthyosis, is slight or transient. On the contrary, eversion caused by paralysis of the orbicularis, chronic inflammation of the conjunctiva or of the edges of the lids, or of the lachrymal ducts, is of a severer degree. Cicatricial contraction of the palpebral border and its vicinity, due to injuries, burns, erosions, gangrene, lupus, anthrax, and syphilitic destruction of the lids and skin of the face, gives rise to true cicatricial ectropion. Mechanical ectropium is due to lack of tissue of the lid, to lid coloboma, and to wounds. Partial cicatricial ectropion which is due to tuberculous osseous affections of the orbital margins and the neighboring osseous parts (zygomatic bone, etc.) is usually situated at the inferior-exterior orbital margin. In this affection the lower lid, or lids, are everted oftener than the upper ones. If the two puncta lacrimalia are simultaneously everted, there will be an overflowing of the tears. If this condition has persisted for a long time, it will give rise to serious swelling and thickening of the conjunctiva (ectropium uxurians or sarcomatosum) and to xerosis of the corneal epithelium, as well as to corneal ulcers. The latter are particularly dangerous. When anæsthesia of the cornea also exists there is probably some paralysis of the trigeminus.

"Ectropium spasticum" develops from spasm of the orbicularis. It occurs in eczematous conjunctival keratitis of childhood, is complicated by photophobia, blepharospasm, more or less pronounced swelling of the conjunctiva and relaxation of the skin of the lids. It may also occur during an attempt to open the lids simply to examine the eye. Then the inner surface of the cedematous and everted lid bulges forward, becomes strangulated, and the ectropion increases in size with increased contraction of the orbicularis. This may happen in coughing, crying, or even in mimicking facial movements. When the conjunctiva is long everted, it often becomes covered with crusts, and in the course of time develops proliferations. The lid opening is then closed. The upper lid is affected in most cases, less often both lids, and the lower lid alone still less frequently.

The *treatment* depends upon the cause. If there is but slight ectropion of the lower lid, it may suffice to pass pointed cotton pledgets over the everted lid from below upward and carry out similar horizontal movements, the lid being slightly pressed against the anterior surface of the bulb. Should the upper lid be affected, the same manipulations are made, but in the opposite direction.

In the initial stages spastic ectropium may sometimes be reduced by drawing the upper lid, for instance, upward, at the same time pushing

the exposed swollen conjunctiva backward, and drawing the palpebral border downward; both eyes are bandaged immediately after this procedure. Goldzieher uses a cocainated ointment on a bandage which is frequently changed. The result is often favorable. If this is not sufficient, a temporary blepharotomy is done; or, under careful aseptic precautions, one or more of Snellen's sutures are inserted from the cul-de-sac to the skin of the lid.

The treatment of paralytic ectropion has been described on p. 104. Cicatricial and mechanical ectropion usually demand operative treatment, and should be left to the ophthalmic surgeon. This is especially the case if there is a cicatricial adhesion of one or both lids to the orbital margin, it being usually necessary to excise the cicatrix and to remedy the defect by a plastic operation.

25. AFFECTIONS OF THE LEVATOR PALPEBRÆ

The upper lid is relaxed in paresis and paralysis of this muscle, causing ptosis (Fig. 1). The congenital form has been described on p. 5. This form of ptosis may be an hysterical manifestation when the oculomotor nerve is affected, or it may be due to the presence of a foreign body in the orbit, or to injuries, such as dissection, tearing, or avulsion of the muscle or its tendon. In childhood it is a rare symptom of myasthenia, or a local manifestation of polymyositis.

Differential Diagnosis.—In ptosis caused by paralysis of the levator muscle, the pupil of the affected eye is usually dilated. Hysterical ptosis has the same external appearance, but is differentiated by the presence of other hysterical disturbances, by the possibility of psychic influence, and by its behavior in involuntary raising of the lid. Hysterico-spastic, pseudoparalytic ptosis is caused by spasm or increased innervation of the orbicularis muscle, and is characterized by considerable nictation and the formation of folds in the skin of the lids. This often follows a tonic spasm of the orbicularis. On the other hand, the symptoms of paralytic ptosis are absent, viz., contraction of the frontal muscle and the arched eyebrow. Finally, resistance is felt on trying to lift the brow. When this is attempted there are twitching of the fibres of the orbicularis and spasmodic contraction of the interior and inferior recti. Sometimes normal curve of the lid is changed to an almost horizontal line.

Pseudoptosis is due to smallness or absence of the globe, to adhesions of the conjunctiva of the upper lid with the bulbar conjunctiva, to inflammatory swelling, tissue infiltration and thickening of the skin of the lids, to callous formations in the upper cul-de-sac of the superior conjunctiva in and after trachoma, or to tumors.

The form of myasthenic ptosis which is often accompanied by paralysis of the palpebral and ocular muscles, as well as by myas-

thenic reaction of the deltoid muscle, has sometimes been erroneously attributed to cerebral syphilis, tabes, and rheumatism.

Von Michel observed in a ten-year-old boy unilateral, hereditary syphilitic parenchymatous nephritis in the course of myasthenia.

In incomplete paralytic ptosis the upper lid moves more slowly than that of the normal eye in upward vision. This is not the case in sympathetic or pseudoptosis. In the congenital form, which is often incomplete, the degree of ptosis is subject to frequent variations. When the patient is excited, the lid opening may even become abnormally wide. As a rule, there are also congenital motor anomalies of the globe—usually upward—and abnormal participation by the upper lid with movements of the eye in certain directions. Even in the acts of mastication and deglutition the globe or lid may make simultaneous movements. But this is rare.

In sympathetic ptosis the upper lid droops moderately. The temporary widening of the lid opening, which can be accomplished in paralytic ptosis by instilling cocaine, will not occur in the sympathetic form. On the other hand, some signs may be present of paralysis of the fibres of the sympathetic nerve in the neck. These consist in a constriction of the lid opening, myosis, hypotony of the eye, slight exophthalmos, unilateral perspiration, and hemiatrophy of the face. If the ciliary ganglion is affected, there are also trophioneurotic manifestations of the eyes themselves and of the lids.

The *treatment* of traumatic ptosis consists in the removal of any possible foreign body from the orbit and suturing of the levator palpebræ. In the other forms of ptosis, also, of course the treatment depends upon the cause. In regard to the constitutional treatment of myasthenia, special attention should be paid to the general muscular condition and to building up the constitution.

As to operations for ptosis, see p. 42. None should be undertaken, unless the affection has persisted for a number of years and is otherwise incurable.

Posey observed an eight-year-old boy with clonic spasms of the levator, resembling chorea, with a rapid and more or less rhythmical raising and lowering of the upper eyelid.

Other concomitant movements of the lid and globe might be mentioned; among these is the inability to lower the upper lid in looking downward, notably in Basedow's disease. This is the v. Graefe's sign. The pseudo-v. Graefe sign consists in abnormal raising and lowering of the upper lid in motor disturbances of the eye. This is found especially in unilateral paralysis of the oculomotor in the stage of involution. Abnormal raising or lowering of the upper lid may accompany adduction, abduction, or convergent and pupillary movements.

Fleischer observed in a hydrocephalic child of one year that the globe during sleep turned downward, disappearing under the lower lid (inverse Bell's phenomenon). In that case the upper lid was also considerably drawn upward by an enormous deformity of the skull, thus producing a gaping of the palpebral opening.

As to the effects which symblepharon may have on the palpebral movement, compare chapter on affections of the conjunctiva (p. 125).

26. TUMORS

Epithelioma contagiosum (molluscum contagiosum) occurs only in childhood. Bollinger attributes it to gregarinæ. Sanfelice thinks it is due to blastomycetes. It usually attacks the lids of one eye or else both alternately, and usually the middle half of the margins of the lid. Other points on the face or the upper extremities may be affected at the same time. Contagion, including that by auto-infection, is a demonstrated fact. Molluscum may also cause secondarily a catarrhal conjunctivitis.

Treatment.—The ulcers are incised and touched with silver nitrate, or snipped off with scissors.

Warts (*verruca plana juvenilis*) are touched with trichloracetic acid and lactic acid. The contents of comedones and of milia are pressed out after the thin epidermal layer has been scratched with a cataract needle (Plate XXI, Fig. 2). Milia are often very numerous on the skin of the lid.

Hæmangioma tuberosum simplex (lymphangioma tuberosum multiplex) is a tumor which may be mistaken for milia or cystadenoma of the lid. It is usually removed for cosmetic reasons.

Soft sarcoma, myxosarcoma, and glioma should be operated upon as early and as radically as possible. The latter frequently recurs after the removal of a gliomatous globe. Pseudoleukæmia, on the other hand, is more suitable for internal treatment with arsenic, etc.

Atheromatous cysts should be carefully and completely evacuated in order to prevent relapses. However, owing to the attenuated walls, this is not always an easy matter.

Injuries to the lid are treated of in the section on injuries to the eye.

V. AFFECTIONS OF THE LACHRYMAL ORGANS

THE two lachrymal glands, like the salivary glands, are doubly supplied with nerves: by the facial as the excitolachrymal nerve, and by the sympathetic nerve. The larger orbital gland is located in a fossa on the temporal side of the orbital roof and therefore cannot be seen. The smaller or accessory palpebral gland lies behind the upper cul-de-sac of the conjunctiva, in the shape of a slightly warty structure. It can be seen near the external canthus by everting the upper lid and drawing it up with the finger, while the eye looks down toward the nose.

The two canaliculi themselves cannot be seen, but the upper and lower puncta may be observed at the inner canthus. The other parts—the lachrymal sac and the nasolachrymal duct—are not visible. The anterior wall of the sac, which is located in the groove of the lachrymal bone, is subject to great variations in size. It connects with the nasopalpebral ligament in such a way that the latter bulges forward as a horizontal crest when the lid opening is drawn toward the temple. This nasopalpebral region normally assumes the shape of a groove. Its flattening or bulging therefore suggests a pathological change of the lachrymal sac. A watery, mucous, or purulent discharge from one or both canaliculi, on pressure, shows an abnormal accumulation of fluid.

Anomalies of the nasolachrymal duct, such as constriction, etc., can be diagnosed only by testing its permeability. If any such anomalies exist, a thorough rhinological examination is indicated, since they may be caused by pathological conditions of the nose and its accessory sinuses. Should this examination fail to give definite data, as is often the case, a probable diagnosis is made of constriction dependent on causes in the canal itself.

The movements of the lids are the principal factors in accelerating the flow of the tears. In exerting that power, they are greatly aided by spontaneous muscular movements of the canaliculi.

It is still uncertain to what degree this function is further aided by dilatation of the lachrymal sac, or by a sort of valvular action of the folds of mucous membrane in the canal— if indeed those are factors of any importance.

But we know that the lachrymal secretion can be decreased even when there is good permeability of the nasolachrymal canal, perhaps because of atony of the lachrymal sac. Possibly a kind of aspiration of the tears takes place in the act of respiration.

It is still a matter of controversy whether the lachrymal fluid has any bactericidal properties.

Onodi's anatomical examinations have established the following important etiologic-pathogenic relations between the nasolachrymal canal and the nose and its accessory sinuses:

The diameter of the nasolachrymal canal was 1.5 mm. in the newborn and in infants of two, four and a quarter, four and a half, five, five and a half, eight, eleven, and twelve months old; it was 2.5 mm. in a three-year-old child. In an infant of five and a half months it communicated with the lower nasal duct by an aperture measuring 2.5 to 3 mm. The narrow lower opening of the nasal duct sometimes found in the first years of life may be due to pressure of the adjacent inferior turbinated bone, as this often touches the bottom of the nasal cavity. It also happens that in childhood the antrum approaches the wall of the nasolachrymal duct, although the duct is separated from the antrum by a spongy osseous layer, varying from 1 to 3 mm. in thickness. As the child grows the antrum enters into progressively closer relations to the duct. Any constriction of the nasal duct favors infection of the entire nasolachrymal duct, as, for instance, in gonorrhœal nasal catarrh, or in suppuration of the accessory sinuses.

1. INFLAMMATION OF THE LACHRYMAL GLANDS

Acute dacryoadenitis may occur as a metastatic sequela of dysentery, spotted fever, measles, scarlet fever, or diphtheria. In diphtheria it is often associated with swelling of the parotid, cervical, and submaxillary glands, and with acute catarrh of the middle ear. One case of measles was followed by considerable swelling of the cervico-sublingual and cervico-submaxillary glands.

Diagnosis.—In acute primary idiopathic or in secondary metastatic inflammation of the lachrymal glands there is painful swelling and erysipelatous reddening of the lids, especially at the temporal part of the upper lid. This is accompanied by local and also by a general elevation of temperature, which, however, does not exceed 100 to 100½° F. The eyeball is displaced downward and toward the nose. Sometimes it is displaced anteriorly, and its motility restricted. The general state of health is either not disturbed, or only to a small extent.

If the upper lid is pressed slightly upward and the globe is at the same time turned downward and toward the nose, then the rough, enlarged, and painful gland at once becomes visible; or it may bulge forward at the transitional fold as a tongue-shaped, œdematous fold. There is often a watery or mucopurulent secretion from the hyperæmic, chemotic conjunctiva. To differentiate dacryoadenitis from blennorrhœa of the conjunctiva, it should be observed that the changes of the skin of the lid and conjunctiva gradually become less pronounced toward the nasal canthus. It differs from circumscribed abscess or periostitis of

the temporal part of the orbital wall, in that the conjunctiva in the latter affection is smooth. In chalazia there is a circumscribed, palpebral infiltration, which is absent in dacryoadenitis.

Spontaneous evacuation usually occurs by perforation of the pus into the conjunctival sac at the upper transitional fold. It empties less often externally through the skin of the upper lid.

CHRONIC DACRYOADENITIS of the unilateral or bilateral variety occurs most frequently after parotitis, but it may also precede this affection. Dacryoadenitis takes an acute or subacute, but rarely a chronic course. Another form of the disease, running a course similar to parotitis, has also been described. This is accompanied by swelling of the sublingual, pre-auricular, and submaxillary salivary and lymph-glands. In a few cases, the lymph-glands of the neck or of the entire body have been involved, but not the parotid. Even tuberculosis of the iris has been described as part of the "symptom-complex of v. Mikulicz disease." In fact, the type assumes many different forms, and has not yet been clearly defined.

In inflammatory or tumor-like enlargements of the lachrymal gland of luetic origin the local symptoms are slight and correspond rather to those of chronic dacryoadenitis. But the lachrymal gland is firmer than in non-specified dacryoadenitis.

The effect of syphilitic remedies influences the diagnosis. Tuberculosis of the lachrymal glands is sometimes bilateral. It may be relatively acute and develop within a month or so, along with ptosis and swelling of the lachrymal glands. At first there may be subjective manifestations (burning pains, etc.); or, the onset may be gradual, the malady extending over several years.

A firm, cartilaginous tumor, about the size of a bean or almond, appears in the upper temporal part of the orbit. As a rule, it cannot be sharply outlined, but it is easily movable, and unattached to the skin, although the latter may be red and swollen. The auriculo-salivary and pre-auricular lymph-glands on the affected side may also be tuberculous. One case was complicated by a papillary proliferation of the injected and swollen conjunctiva, and pannus of the cornea.

In the differential diagnosis the history must be considered, and also any previous catarrhal inflammation of the conjunctiva, sarcoma of the orbit, and any injuries in the orbital, frontal, or temporal regions.

In acute and chronic conjunctivitis the lachrymal glands are rarely involved.

Treatment.—In acute dacryoadenitis, ice-bags or cold compresses may be tried first. If fluctuation is present an incision is made from without about $\frac{1}{2}$ inch long, parallel to the temporal orbital margin, and close to it, so as to protect the orbital structures, such as the tendon of the levator.

After that a fresh gauze tampon should be introduced daily until the purulent discharge has been arrested. The incision usually heals satisfactorily under a thin layer of a 1 per cent. yellow oxide of mercury salve, spread on borated lint.

In subacute dacryoadenitis cooling lotions assist in relieving pain and in the improvement usually effected in a few weeks. Internally, calomel and potassium iodide are useful to facilitate absorption.

The chronic type is treated by local massage and by painting the palpebral skin with tincture of iodine. Internally, syrup of the iodide of iron, arsenious iodine, and Fowler's solution are administered, and externally blue ointment is used in the usual doses. In tuberculosis of the lachrymal gland it is extirpated from without by incision of the skin at the superior orbital margin. The same course is indicated for new-growths. These are usually mixed tumors. Removal of part of the orbital wall or resection of the temporal orbital wall may be necessary, as advised by Kroenlein.

The cystoid dilatations of the excretory ducts are caused by concretions or adhesions, or by cysts of the palpebral part of the lachrymal gland. This was observed by Dor in an infant of three and a half months. Such a condition requires excision through the part which is most easily reached. Compare also the chapter on "Injuries to the Eye."

2. PARTIAL OR TOTAL ARREST OF LACHRYMATION

This occurs as a purely nervous manifestation in Asiatic cholera or in the vomiting and purging of dysentery. It is then the result of the closing of the excretory ducts of the lachrymal glands. It also occurs in atrophy of the lachrymal glands following paralysis of the trigeminus or facial nerve, in partial paralysis of the sympathetic, and in exophthalmos. The object of the treatment in cholera, cholera, and dysentery is to reduce the evaporation from the globe by warm boric acid compresses, and to prevent ocular infection by instillation of salicylic physostigmine. The dry sensation is diminished by instillations of freshly-boiled milk. In the form of the affection which is caused by atrophy of the glands, protective spectacles are useful.

3. EPIPHORA (PATHOLOGICAL LACHRYMATION)

This affection is due rather to an obstruction interfering with the excretion than to exaggerated function of the lachrymal glands. The simultaneous occurrence of both causes is an exception. Reflex hypersecretion is usually caused by irritation of the trigeminus in the eye or mucous membrane of the nostril, by injuries to the conjunctiva or cornea (foreign bodies, etc.), inflammatory changes of the eyeball and its

vicinity, affections of the nose, and neuralgia of the trigeminus. Epiphora may also be caused, even in healthy ducts, by dilatation of the antrum, inflammation of the dental roots, by a sharp wind, or by impurities in the air or too strong light. This may possibly be due to irritation of the fibres of the trigeminus. According to Weleminsky, non-obstructive epiphora may be due to hypertrophy of the anterior end of the middle turbinate, since irritation of that place causes exceedingly strong lachrymation. Application of cocaine exacerbates this condition, while scarification improves it.

Labyrinthal epiphora, which is regarded as a reflex manifestation, is rare. In a soldier, aged twenty-one, who suffered from bilateral catarrh of the tubes, Cornet observed that lachrymation would occur with conjunctival injection as soon as the right manubrium was moved.

In lachrymation accompanying tabes and Basedow's disease there are no pathological changes of the conjunctiva or nose. The eyes weep in the open air, especially when the weather is cold.

Purely mechanical obstruction of the lachrymal and nasal secretion may be caused by congenital absence of the puncta lacrimalia and canaliculi, by impairment of the physiological lid movements, such as paralysis of the facial nerve, by eversion of the lower punctum, by notches or coloboma of the lid, shortening of the lids, paralytic and cicatricial ectropium, by foreign bodies in the puncta lacrimalia and canaliculi, or by similar mechanical impediments in the lachrymal vessels. Empyema of the accessory sinuses, which is nearly always associated with swelling of the turbinate, interferes only indirectly with lachrymal secretion, or through the nasal catarrh spreading to the duct. It is a noteworthy diagnostic point in unilateral disturbances of this kind that patients often complain not only of lachrymation, but also of xerosis and occlusion, or of greater moistness of the corresponding nasal passage.

As to differential diagnosis, an epiphora, or a chronic ulcerative blepharitis and conjunctivitis in one eye only suggests an affection of the excretory lachrymal vessels of the same side.

Affections of the lachrymal canal and conjunctiva may also be due to a tray-shaped lower turbinate, to diffuse hypertrophic rhinitis with uniform enlargement of the corpora cavernosa, to circumscribed papillary hypertrophy, myxomatous degeneration of the anterior end or vesicular swelling of the lower turbinate. A pronounced deviation of the septum will act as an obstruction, as will a circumscribed periostitis near the ostium, adhesion of the turbinate to the nasal floor, or upward bulging of the latter, owing to teeth perforating into the nasal cavity.

Cystic dilatation of the sac, caused by these displacements of the nasal opening, may disappear after removal of the obstruction, but in most cases they will lead to erosions, ulcers, and scars, both in the canal and sac.

Any inflammatory affections of the nose and adnexa may spread by continuity and contiguity to the nasolachrymal duct. Among these affections are acute coryza, nervous (vasomotor) coryza, chronic diffuse purulent rhinitis, ozæna, adenoid vegetations, etc.

According to Kuhnt, infectious secretion, finding its way into the nasolachrymal sac when blowing the nose, may cause any of the phlegmonous inflammations of the lachrymal sac, including probably chronic dacryocystitis and "air tumors" of the sac.

The *treatment* of "hypersecretory" lachrymation is in the first place directed to the removal of the cause, whether general or local. In reflex lachrymation, proper glasses should be prescribed, especially if the vision is impaired.

One of my favorite plans is to use a weak astringent lotion in addition to any of the other local methods advised.

This palliative treatment is also useful in congenital absence of the lachrymal puncta and canaliculi, and in atresia of the canaliculi which follows trachoma or injuries. It can also be used with benefit in incurable extensive cicatricial ectropion of the lids.

The removal from the lachrymal puncta and canaliculi of foreign bodies, such as visible cilia, bristles, portions of scalp hair, particles of wood, seeds, and husks, has already been indicated.

Fungous concretions (*Streptothrix actinomyces*) when walled off are absorbed as a slowly developing affection, located, as a rule, in the lower canaliculus, although they sometimes appear in the upper one. Here the colonies rapidly undergo degeneration. The concretion gives rise to an ulcerative process in the surrounding tissue. Actinomyces of the nasolachrymal duct has been described.

In the *differential diagnosis* of other forms of lachrymation several points are important. First is the history. We should look for such as the following: redness and catarrhal swelling of the conjunctiva in the vicinity of the canaliculus; an elastic, semispherical prominence at that place, over which the skin may be normal and movable, or redness of the corresponding part of the edge of the lid; absence of simultaneous affection of the nose; lachrymation of one eye only; infection from corn husks. The corresponding punctum may be occluded or dilated to several times its normal diameter, or it may be everted. Probing the canaliculi, preferably with a blunt conical sound, will show the occlusion or dilatation, or it may lead into a space filled with soft granular tissue covered with a yellowish-white fluid. Occasionally it happens that a more or less bad-smelling mass will be evacuated outward, showing the presence of staphylococci and the *Bacillus mensesentericus ruber*.

The microscopic demonstration of the mycelia by Gram's method is always necessary, although, because of the slow development of acti-

nomycosis, the staining is not always distinct. This is true also of the clinical picture, if the inferior punctum and canaliculus are displaced downward by streptothrix. The upper canaliculus is seldom involved. There is no doubt that these affections are often mistaken for each other. According to Axenfeld, only one type of cases is due to actinomycosis; in the others various kinds of streptothrix must be considered—never leptothrix and kladothrix. One patient was in the habit of moistening his lids with saliva.

Treatment.—I have effected cures with preservation of the canaliculus by gradually dilating the lower canaliculus with conical sounds of varying diameters (Plate XXI, Fig. 5) and injections through the upper one of a solution composed of equal parts of bichloride, 1 in 10,000, distilled water and glycerine, gradually increasing the force of the piston. The injected water, when passing out through the lower canaliculus, loosened the accumulations located at the entrance to the lachrymal sac. Then pressure upon the sac and the neighboring parts of the lower canaliculus with a Daviel's spoon caused the entire contents to be evacuated through the lower punctum. Having repeated the injection, I carefully cauterized the bottom of the cavity with silver nitrate fused to the point of a conical sound.

If this plan cannot be followed, the canaliculi should be incised. Loewenstein observed a concretum of hypomycetes in an inflamed extirpated lachrymal sac.

Where there is a tendency to perforation outward, an incision should be made parallel to the border of the lid.

If the sac and nasolachrymal canal are likely to be involved, the former should be incised or removed. It may also become necessary to scrape the nasolachrymal canal with a bent spoon, grooved at the back.

Epiphora.—This often depends upon a slight eversion of the lower punctum, as, for instance, in paresis of the facial nerve; or it may be a sequel to scars from injuries or smallpox at the lids and vicinity; or it may occur in chronic blepharoconjunctivitis. The correct position of the puncta lacrimalia is tested by closing the eyes gently, when the puncta should meet with such exactitude that the lachrymal sac and canaliculi form a perfectly closed canal. But if, on gently lifting the nasal part of the palpebral fissure, or on having the patient incline his head forward and look upward, the puncta deviate ever so slightly, then the normal drainage of the lachrymal fluid is interfered with. When this anomaly and eversion of the puncta and canaliculi are unilateral, they will be recognized by comparison with the healthy eye.

Unless the eversion is corrected, wiping of the eyes will produce an increasing ectropion.

Should treatment of the causes prove unsatisfactory, the condition

may still be improved by incising the affected canaliculus and keeping it open for three or four days with a conical sound, or by excising a triangular piece of the palpebral conjunctiva at the punctum itself. The resultant shortening of the tarsal conjunctiva may cause the punctum to return to the lacus lacrimalis.

As unusual complications may be encountered, even these operations should be left to the specialist.

When neither the puncta nor canaliculi are constricted or occluded, the question arises whether there is any mechanical obstruction in the lachrymal canals. This is tested by pressing with the finger over the region of the lachrymal sac, taking care not to compress the puncta or canaliculi; also by irrigating the lachrymal sac and nasolachrymal canal. This is most conveniently effected from the lower punctum. The insertion of an Anel's syringe (Plate XXI, Fig. 7) for this purpose is facilitated by previously instilling alypin-suprarenin into the punctum and canaliculus. The course of the latter is changed from an angular to a straight direction by placing a finger of the left hand at the lower edge of the lid and then making traction down and outward. The canaliculus can then be dilated with a fine conical sound. In order not to injure the punctum and canaliculus, the sound is introduced slowly in corkscrew fashion until it has arrived at the interior nasal wall of the lachrymal sac. Tension of the lower lid is then effected, and the syringe, filled with a tepid, sterile, 0.9 per cent. solution of sodium chloride, is inserted vertically into the punctum, after which it is horizontally advanced into the canaliculus as far as possible. By exerting a uniformly gentle and gradually increasing pressure upon the piston, injury to the tissues will be avoided. Under normal conditions the irrigation will proceed smoothly. The patient inclines his head slightly forward, so as to prevent the water from trickling into his throat.

A still gentler way of testing the permeability of the lachrymal canal is by dropping a 2 per cent. solution of fluorescein-potassium into the conjunctival sac (Dalen). After repeatedly and forcibly closing the lids the fluid will often appear at the nostril of the same side, if conditions are normal. Schirmer recommends repeated instillations of sodium salicylate, continuous movements of the lids, and then blowing the nose into a cotton pad which has been coated with a 1 per cent. solution of ferrum sesquichlorate. If the test is positive, the pad will soon assume a violet to brownish-red tint; otherwise the discoloration is delayed or absent. The instillation test, however, has the drawback that it may prove negative even if the lachrymal sac and nasolachrymal canal are quite normal.

Strictures in the lachrymal sac and duct are demonstrated by introducing the finest number of Bowman's sounds (Plate XXI, Fig. 6) through the unincised but dilated canaliculus into the nose.

General practitioners should leave this process to the specialist, as even they may not always be able to avoid injuries to the lining membrane. These may, in turn, lead to constriction and obliteration of the puncta and canaliculi with subsequent epiphora, and necessitate dilating the puncta and canaliculi by sounds of increasing calibre.

4. BLENNORRHOEA OF THE LACHRYMAL CANALS

Blennorrhœa occurring during an affection of the lachrymal sac, either in a lingering blepharoadenitis or as a primary affection, is recognized by the puffed eyelids in the region of the canaliculi, protrusion of the lachrymal puncta, gaping of the canaliculi, and bulging of their mucosa. Granuloma in the canaliculi has also been observed.

Treatment.—In fresh cases the canaliculi should be frequently expressed and irrigated with oxalycyanate (1 : 5000), and in cases of free secretion with a 1 per cent. solution of silver nitrate. If there is considerable protrusion and if this ectasia occurs after extirpation of the lachrymal sac, or as a trachomatous affection of the mucosa of the canaliculus, the latter should be scraped with a small sharp spoon and touched with a 2 per cent. solution of silver nitrate.

5. ACUTE (PHLEGMONOUS) DACRYOCYSTITIS AND BLENNORRHOEA OF THE LACHRYMAL SAC (PLATE II, FIG. 5)

Swelling of the mucous membrane of the nose in scarlet fever and in catarrhal influenza seems to favor the development of acute suppurative dacryocystitis. This is especially the case if there exists already a chronic rhinitis with constriction of the nasolachrymal canal, or a chronic inflammation of the lachrymal sac. There is also a tendency with this to relapses.

An acute dacryocystitis (pericystitis lacrimalis) is sometimes only the local expression of an erysipelas or of a streptococcus infection.

Typhoid bacilli in pure culture have been found in acute post-typhoid dacryocystitis in an eleven-year-old child.

Like the mucous membrane of the nose, that of the nasolachrymal canal may be covered with smallpox pustules, so that in the florid stage acute dacryocystitis, resulting from chronic catarrh of the lachrymal sac and post-variolar blennorrhœa of the lachrymal sac, is not rare. They may also be caused by secondary changes of the mucous membrane of the nose and lachrymal canal.

Dacryocystitis sometimes results from the spreading of a variolophlegmonous inflammation of the skin above the lachrymal sac. Or, variolar pustules in the vicinity may produce constriction, occlusion, or displacement outward of one or both puncta.

The lachrymal secretion is very rarely involved in gonorrhœal or diplococcus infection of the conjunctiva.

Diagnosis.—Acute phlegmonous dacryocystitis is usually unilateral. Its external manifestations begin with reddening and swelling of the overlying skin of the lachrymal sac, which may spread to the neighboring parts of the lid and conjunctiva. The region is painful to the touch, and there is often swelling of the pre-auricular and submaxillary lymph-glands, disturbed sleep, and elevation of temperature. As the swelling increases, the skin soon becomes thin and yellow. The pus perforates outward. Usually the opening is below the edge of the lid, or toward the conjunctival sac. Perforation into the orbit may occur if there is chronic dacryocystitis and unusual thinness of the orbital septum. If the pus enters the adjacent sinuses, the globe protrudes outward and obliquely down or up, according to the lesion. Cloudiness and secondary ulceration of the cornea may also occur. Sometimes, while the phlegmons are still in a state of involution, optic neuritis may develop as the result of the attending induration, which, in turn, may lead to atrophy of the optic nerve and persistent motor disturbances. The internal and superior recti and the levator palpebrarum are especially likely to be involved. Fatal meningitis has also occurred.

Chronic dacryocystitis (dacryocysto-blennorrhœa) is a much more frequent affection and is often bilateral. It is distinguished by the presence of highly-virulent pneumococci. There are no such rapid pathologic changes as occur in the acute form, but there may be much lachrymation, sticking together of the cilia or of the palpebral borders, due to a mucopurulent secretion, and swelling in the region of the lachrymal sac. The secretion, however, may have been already pressed out by the patient, or may have escaped to neighboring parts of the nasal cavity, so that even pressure on the sac will not cause any discharge. Even if the puncta are in position, it is important to establish whether there is any serious obstacle in the secretory lachrymal vessels, and whether the sac has been invaded. This can be done by injecting a fluid into the canaliculus, after dilatation with a sound. Under normal conditions, the injected fluid simply flows through into the nose and throat. If the canal is obstructed by cicatricial strictures there will then be a puffing out of the sac. Such cicatrices result from previous ulcers, which are usually situated at the neck of the sac, or by occlusion of the entire canal.

Polypous proliferation of the mucous membrane of the sac is not rare, in cases where the inflammation is of a protracted character.

It is important in differential diagnosis to note that the swelling in dacryocystitis extends only slightly upward above the ligamentum palpebrarum, while in empyema of the frontal cavity it is particularly noticeable upward and above that ligament. The relation to the affections of the accessory sinuses, mentioned on p. 110, should also be in-

quired into. Thus, an ectasia of the cribriform cells and of the lower wall of the frontal cavity may considerably compress the upper part of the lachrymal sac, simulating dacryocystitis. Again, empyema of the accessory sinuses, especially of the cribriform cells, may spread by a subperiosteal abscess, perforating into the sac, or by perforation of the sac (pyosaccus). Empyema of the maxillary cavity damages the osseous wall of the duct, especially in the region of the recessus prælacrimalis, by occluding the lumen of the duct. This may happen through abrasions, holes, and sequestration, or by periosteal proliferation.

The sooner dacryocystitis is recognized, the better, not only on account of the annoying ulcerous blepharitis which often prevails, and the eversion of the lower lid, but particularly because if there is the least injury to the cornea, the contents of the lachrymal sac very often cause a serpiginous ulcer of the cornea. Even in favorable cases this nearly always leads to diminished visual acuity of the affected eye.

Tuberculosis of the nasolachrymal duct is almost always caused by lupus or by tuberculosis of the nose, lids, or conjunctiva. Lupotuberculous dacryocystitis is accompanied by doughy distention and thickening of the region of the lachrymal sac and outward perforation of discolored granulations. The skin above the lachrymal sac may undergo ulcerous disintegration, spreading beyond the borders of the sac to the skin of the lower lid. In other cases the skin over the sac is not changed at first. Lupus nodules penetrate into the deeper parts, causing degeneration of the mucosa of the sac. At the same time, the lachrymal duct often remains permeable for irrigation, or the suppuration of the sac in nasal lupus may be simply due to stagnation of secretion.

Tuberculous ulcers of the mucosa, and tuberculous caries of the osseous nasolachrymal canal and the lachrymal bone, which occur relatively often in infancy and childhood, are suggested by the presence of rough bone during sounding, and simultaneous nasal tuberculosis and skin fistulæ of the lachrymal sac, which will persist after removal of the sac.

The *local treatment* consists in removing the pathological tissue with the sharp spoon and tamponading with iodoform gauze.

Syphilitic affections of the nasolachrymal canal and sac, catarrh, blennorrhœa, constriction amounting to impermeability, phlegmonous inflammation, fistulæ or destruction of the mucosa, are always spread from inflammatory ulcerous affections of the nasal mucosa, or from ostitic and periostitic swellings, and necrotic destruction of the neighboring bones.

The syphilitic affections in the nasal end of the lids and conjunctiva, of the lachrymal puncta and canals, are always caused by disturbances of this kind. Primary carcinoma of the lachrymal sac is rare,

while secondary trachomatous infection of the sac and the nasolachrymal duct occurs oftener in the wake of a conjunctival trachoma. However, dacryocystitis in trachoma is not always specific.

Treatment.—In acute dacryocystitis, moist warm compresses may effect a cure. In most cases, however, abscess formation is unavoidable, especially in phlegmonous dacryocystitis. Maturing is enhanced by cataplasms, moist or dry heat. If the abscess perforates outward, this treatment is continued until the cure is complete. The resulting scar is so slight and smooth that it will hardly be noticed. If, however, there is an impending perforation into the deep parts, the abscess should be opened, as follows: The lids are carefully closed, and ethyl chloride is sprayed on until the skin over the lachrymal sac turns white. The anterior wall of the sac is then perforated with a bistoury directly below the centre of the inner palpebral ligament, the back of the knife being directed upward and inward, the edge downward and outward. The inner palpebral ligament is then exposed by drawing the lid fissure outward with the thumb of the disengaged hand. Any necrotic skin shreds are removed, and the ulcerated tissue gently scraped away with a sharp spoon. The wound is cleansed with cotton tips, and a strip of sterile gauze is inserted into the sac and renewed daily, until the reactive manifestations disappear and there is no more secretion from the mucosa of the sac. Finally, a moist bandage is applied. Should there be much swelling and catarrh of the conjunctiva, the moist bandage is replaced by a sterilized piece of soft gauze, which is fastened with elastic collodion to the skin surrounding the wound. The patient should close his eyes so as to prevent the collodion entering the conjunctival sac.

If the wound is slow in closing, or if a fistula of the lachrymal sac has developed following spontaneous rupture, the traumatic margins should be touched with argentic nitrochloratum.

A constriction of the nasolachrymal canal is usually situated at the junction of the sac and the canal, or at the lower end of the latter. It is treated by carefully inserting a thin sound through the wound into the nasolachrymal canal every few days, gradually increasing the diameter of the instrument. It is allowed to remain in place from five to fifteen minutes and then withdrawn slowly. The sound is advanced in the sac until its point touches the inner wall, and is then raised to a nearly vertical position, so that its lower end in the lachrymal sac points to the sulcus between the nasal alæ and the cheek. This indicates the position of the lower ostium of the nasolachrymal canal. The sound is now carefully advanced downward until it touches the floor of the nasal cavity.

Should there be an orbital phlegmon also, it is incised at the lids, or the perforation is dilated. The orbital abscess cavity is then irrigated daily, preferably with freshly-sterilized physiological salt solution, and

loosely tamponaded. Further experience is needed to determine the value of anti-ferment treatment (Lenz).

Fuchs opens the lachrymal sac immediately preceding an iridectomy and cataract operation, if there is a suspicion that it is not normal. He then irrigates and tamponades it with iodoform gauze, which is removed after the bulbar wound has healed. The sac returns to its original condition after the skin wound has healed.

Should there be relapses of dacryocystitis, it may be necessary to eliminate the lachrymal sac in order to prevent the danger resulting from renewed or prolonged stagnation of the secretion in the sac (serpiginous ulcer of the cornea).

I do not consider the introduction of sounds through the previously-incised upper and lower canaliculi in chronic dacryocysto-blennorrhœa good practice. Aside from the impairment of the flow of secretion by changing the tubules into grooves, there is no safe halting place in using the sound. Injuries to the mucosa of the nasolachrymal duct will occur even with the most careful procedure, and they are particularly prone to happen if the osseous nasal canal is constricted by periostitic thickening and exostoses. Polyps in the lachrymal sac may also be caused.

I have repeatedly observed a return of the stenosis after this procedure, amounting to complete impermeability of the nasolachrymal canal. There has even been fatal meningitis due to rupture of the canaliculus without any clinical signs of the orbital tissue being involved. Valude observed atrophy of the optic nerve and amblyopia following circumscribed inflammation of the orbital tissue, after the above procedure had been instituted.

I have observed the following pathological course in a patient with extensive cicatricial entropion and a minute fistula of the lachrymal sac, who had been treated with sounds by an ophthalmologist: three days later there was œdematous swelling of the lower lid, especially toward the nasal region, and hyperæmia of the disc with rigid pupil, which terminated in neuritic atrophy and blindness.

It is for such reasons that I cannot look with favor upon the widely prevailing method of splitting the strictures with a bent button knife, inserted like a sound through the previously-incised upper or lower canaliculus. My practice, therefore, is not to incise the canaliculi, except when there is such a constriction at the puncta lacrimalia, the canaliculi or their communication with the lachrymal sac as to prevent dilatation by the sound. The agglutination of the wound is prevented by introducing a conical sound daily, for several days, into the incised canaliculus.

Before removing a stricture, it is important to have a thorough rhinological examination, as rhinological and general treatment may demand precedence over direct treatment of the eyes.

Catarrhal swelling of the mucosa of the nasolachrymal canal may be removed by very gentle irrigations with different solutions, such as 3 per cent. boric acid, or, if indicated, with sublimate (1 : 10,000), with pyocyanase or zinc. sulph. (0.3 aq. dest. and glycerine $\bar{a}\bar{a}$ 50). These irrigations are made from the lower canaliculus, with the patient in the recumbent position. Syringing the lachrymal sac with equal parts of cocain. mur. 0.5 per cent. and suprarenin (1 : 10,000) in equal parts of distilled water and glycerine often reduces the swelling of the mucosa and increases the lumen of the canal. To prevent toxic manifestations, the patient is cautioned to refrain from swallowing. Immediately after the irrigation, he sits up and gargles with tepid water. Flooding of the conjunctival sac is prevented by the patient gently closing his eyes after the nozzle of the syringe has been introduced into the canaliculus, which has been carefully dilated by a conical sound.

Stronger solutions which affect the delicate tissues, or those that are unreliable as to consistency or stability, should be avoided. Energetic irrigation may burst the wall, allowing pathogenic material to enter into neighboring tissue. Lewis, for instance, reported a case of orbital inflammation following injection of protargol into the sac.

Patients should frequently compress the lachrymal sac with a sterilized cotton tip. Tears should not be wiped off with the fingers or handkerchief, but with a small piece of sterilized absorbent material.

If the canal is occluded by a cause which cannot be removed by local or general treatment, or if the mucopurulent secretion continues, the lachrymal sac should be methodically evacuated several times daily by massage. Patients can be easily taught how to do this. It may have to be continued for many months.

If all these methods prove unsuccessful, the lachrymal sac should be enucleated. This is indicated in chronic atrophy of the mucosa, and in ectasia and atony of the sac (hydrops sacci lacrimalis). Or, if the conical sound, when introduced into the canaliculus, meets with rough or abraded places on the lachrymal bone, denuded of mucous membrane, enucleation is to be resorted to if internal treatment has failed. Such treatment should consist of iodine (potassium iodide 5, spartein. sulph. 0.3, aq. dest. 150, a teaspoonful to be taken once or twice daily in a cup of hot milk), as syphilis is often a contributing factor. It may also be necessary thoroughly to scrape out the bone.

Certain methods of treating chronic inflammation of the lachrymal sac, which belong to the specialist, are still *sub judice*. Among these are permanent sounds, soluble bougies, dilatation of the lachrymal canal with a bayonet-shaped needle 2 mm. thick, as practised by Ziegler, permanent drainage of the lachrymal vessels, dacryocystorhinostomy after Toti. By the latter method, the restoration of spontaneous lachry-

mation is effected by removing a part of the lachrymal bone and the frontal process of the superior maxilla; it might, therefore, be considered in congenital atresia of the lachrymal duct, the sequelæ of which cannot otherwise be effaced.

A form of lachrymation which is usually of little consequence occurs in occlusion of the excretory ducts of the lachrymal glands and in chronic catarrh of the conjunctiva, which is of frequent occurrence in individuals who are suffering from troubles of the lachrymal sac. A palliative remedy consists in instillation of a $\frac{1}{2}$ per cent. cocaine solution, but to counteract its dilating effect upon the pupils a few drops of a $\frac{1}{10}$ per cent. solution of morphin. muriat. gtt. iii, aq. dest. 8.0, glycerin. puriss. 2.0, should be added.

Chronic conjunctivitis and ulcerous blepharitis as sequelæ are prevented by carefully greasing the outer part of the lower lid with borolanolin-vaseline, morning and evening.

Removal of the lachrymal gland should be considered only when the overflowing defies all treatment. But Fuchs is in the habit of doing this immediately after extirpation of the lachrymal sac.

Reduction in the size of the sac by electrolysis, which I have practised for some time, is a simpler procedure.

It is done with a double needle, inserted into the lower gland from the fornix of the conjunctiva, after the conjunctiva has been anæsthetized. The gland is brought to view by eversion of the temporal part of the upper lid, or by means of a blunt double hook. The current is closed and slowly increased to 4-5 Ma.; after two minutes it is slowly reduced to 0, and the double needle withdrawn. Lachrymation will be increased for the first few days, but gradually becomes less, and finally is completely arrested. If necessary, the procedure is repeated once or twice at weekly intervals.

No ill-effects have as yet followed the destruction of the lachrymal gland. Evidently, the accessory glands are abundantly able to supply moisture to the conjunctiva and cornea.

VI. AFFECTIONS OF THE CONJUNCTIVA

THE normal conjunctiva has a moist lustre and is so transparent that through it may be seen the yellowish-white tarsus with its vertical, yellowish-white meibomian glands, the white sclera, which, in youth, has often a bluish tint, and the larger bluish-red vessels running to the fornix and episclera.

Most of the conjunctiva is almost colorless, but at its tarsal edge, where there are delicate and somewhat fuller vessels, it is usually of light yellow or light red.

The larger vessels run to the tarsal edge and there dissolve in the delicate ramifications. These can be seen at the tarsal edge as red, somewhat protruding and slightly tortuous stripes. The surface of the conjunctiva is smooth, except at the caruncle. On the plica semilunaris may be seen small, roundish nodules, and also toward the canthus, where it becomes slightly uneven and of velvet-like appearance. The palpebral (tarsal) conjunctiva is immovable. The fornix is connected with the base by a very loose tissue, so that it may be raised in folds. At the place where the conjunctiva folds back from the lids to the globe there are semicircular folds, one at the lower and one at the upper end. These are smoothed out on closing the lids. They allow the globe to move in different directions, and they also admit of eversion of the lid for inspection of the conjunctiva, and of drawing it away from the globe for a certain distance.

From a therapeutical point of view the following changes of the conjunctiva are important for diagnosis:

Inflammatory swelling of the tissue in the shape of increased folding and puffiness of the conjunctiva; reduction in size or abrasion of the conjunctival sac by contraction of the conjunctiva, or by partial or total symblepharon; the various forms of pathological secretion of the mucosa, and foreign bodies. Small foreign bodies are often drawn into the subtarsal or transitional fold (Plate X, Fig. 2).

Reticular injection points to increased engorgement of the large and medium-sized vessels (Plate III, Fig. 1); uniform, diffuse redness of the conjunctiva indicates plethora of even the smallest vessels. A light red color points to inflammatory injection, a more or less dusky violet hue to stagnation. As to the various kinds of reactive redness of the conjunctiva, according to whether the affection is in the conjunctiva, sclera, cornea, or the eyeball itself, see remarks on p. 193.

The conjunctiva is dark red in orbital hemorrhage, due to injuries, or in spontaneous blood effusions in the fornix and bulbar conjunctiva.

A rusty brown extravasation with yellowish-red or yellowish borders points to a former hemorrhage. In icterus, the conjunctiva is yellow, brownish-yellow, or greenish-yellow. In Addison's disease, in local and general argyrosis, it is dirty gray-brown. In general anæmia it is pale. In spring catarrh it looks as if it were covered with a thin layer of milk with pavement-like proliferations at the upper lid. In follicular catarrh and trachoma the infiltrations appear yellowish, mottled, or red-gray, and are roundish, oval, or confluent in shape. Small hordeoli, chalazia, and infarcts of the meibomian glands appear as yellow-white, yellow, or grayish-yellow dots and patches. Eczematous phlyctenulæ and pustules of the bulbar conjunctiva appear as distinctly protruding whitish, yellowish-white, or yellowish-red spots; the scars appear as bluish-white or tendon-white spots, cords, or nets. Angioma has the appearance of convolutes of dilated vessels. Diffuse redness and inflammatory small-celled infiltration of the conjunctiva, swelling of the papilla of the tarsal conjunctiva, new growths or epithelial, incrassations and proliferations render the ducts of the meibomian glands, the sclera and its vessels indistinct or invisible. As a consequence, the surface of the conjunctiva may lose some of its lustre and look velvety or delicately reticular. It loses its lustre entirely by diphtherial or artificial coating, exudative membranes, yellowish, tough, or half-dried mucus. It may also look dry, as if covered with minute scales or with epidermis (xerosis).

Exudates or pyogenic foci, vesicles, cysts, dilatation of the lymph-spaces and new growths of the mucosa are responsible for elevations of all kinds. On the other hand, there may be excavations due to injuries or to trachomatous, tuberculous, lupous, and carcinomatous ulcers. Partial thickening of the conjunctiva may be due to epithelial proliferation, to inflammatory plastic infiltration and tissue proliferation, or to local—less often general—pure or chemotic œdema. There may also be changes of the surface in the shape of protrusions in all sections of the conjunctival sac, caused by neighboring processes, such as syphilitic tarsitis, abscesses of the lid, etc. The sequelæ of injuries are sometimes confined to the conjunctiva, but they often involve the lids and eyeballs. There may also be abnormal adhesion of the bulbar conjunctiva to the cornea (true and false pterygium), and changes in movability (symblepharon) caused by cicatricial shortening, or by contraction and adhesion of the tarsal to the bulbar conjunctiva and cornea, as the result of injury or disease.

Transitory elevation of the blood-pressure is responsible for hemorrhages in and under the conjunctiva, as well as under the skin of the lids. It may be caused by too high or too low temperature of a bath, the lifting of heavy objects, stooping, or a fatiguing cough—notably in pertussis. The spontaneous hemorrhages in diabetes, cardiac insufficiency, and emphysema are likewise of importance in diagnosis.

PLATE III.

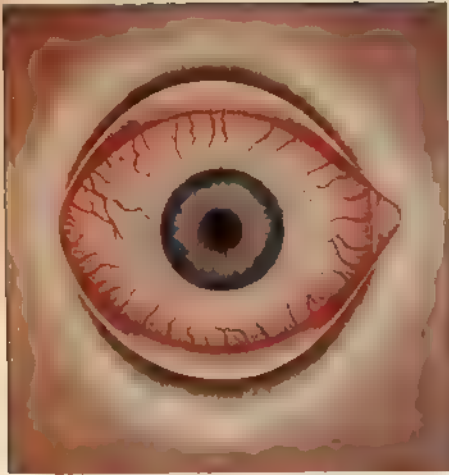


FIG 1 Conjunctival injection

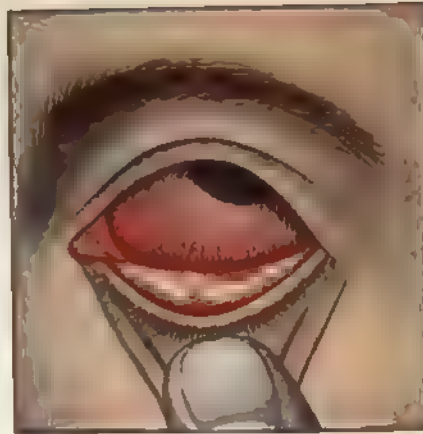


FIG 2 Slough after lineal cauterization of the transitional fold.



FIG 3 Effect of a 5 per cent diamin instillation



FIG 4 Ecchymosis of the conjunctiva in pertussis

A rarer occurrence in childhood is chronic venous conjunctival hyperæmia, associated with dilatation of the facial vessels. It may be due to emphysema, chronic bronchitis, pulmonary cirrhosis, congenital and acquired uncompensated cardiac defects, or to cervical ulcers. If these affections of the heart and lungs lead to pronounced hydrops, œdema of the lids is often accompanied by œdematous swelling and increased secretion of the conjunctiva, as well as slight exophthalmos.

1. HYPERÆMIA AND CATARRH OF THE CONJUNCTIVA (PLATE III, FIG. 1)

At birth the normal conjunctiva is free from germs. Thereafter, the most frequent bacilli present are xerosis bacilli (Plate IV, Fig. 3) and staphylococci. Grave febrile, general affections, such as typhoid and anæmia, do not seem to influence the quantity of infectious germs of the normal conjunctiva.

The bacteriological examination of the conjunctival secretion is increasing in importance in infectious diseases of the conjunctiva, especially from a prophylactic point of view. This is particularly true of acute epidemic conjunctival affections and gonorrhœal infection.

The incubating and ascending stages are the best for examination, and later the climax of the infection. The conjunctival secretion for a dry spread preparation of the cover-glass should be obtained with an incandescent platinum loop and, if at all possible, from the region of the upper transitional fold. Flakes from the mucosal secretion of the lower part of the conjunctival sac are less serviceable. The secretion at the inner canthus is usually polluted by staphylococci and xerosis bacilli. There must be no washing-out of the eye or instillations previous to obtaining the secretion. The latter is spread thinly and uniformly on a well-cleansed slide and, when slightly dry, is drawn three times through the flame, the secretion upward, avoiding too intense a heat. It is then stained.

After fuchsin and methylene blue, Gram's well-known method comes in for consideration. "Gram-positive," meaning blue, will stain most sarcinæ, staphylococci, and streptococci (occurring in streptococcus conjunctivitis); pneumococcus (Plate IV, Fig. 2) occurring in pus from the lachrymal sac, acute pneumococcus conjunctivitis, serpiginous ulcer of the cornea; bacilli of the diphtheria group (xerosis bacilli, pseudodiphtheria, and diphtheria bacilli), bacillus refringens; aspergillus fumigatus; streptothrix and actinomyces. "Gram-negative," meaning red, stains: Koch-Weeks's bacillus (in acute conjunctivitis, which occurs most frequently and is very contagious); influenza bacillus (in conjunctivitis and pus from the lachrymal sac); diplobacillus (Plate IV, Fig. 1; in chronic conjunctivitis and corneal ulcer); colon bacillus (often in acute catarrh of the new-born); gonococcus (Plate IV, Fig. 4); meningococcus,

micrococcus catarrhalis, *bacillus pyocyaneus* (sometimes in very grave corneal ulcers); *pneumonia bacillus* Friedländer (often in the pus of the lachrymal sac).

Bacillus subtilis, which has sometimes been found in very grave panophthalmia, is stained partly Gram-positive and partly Gram-negative. Cultural examination may also be necessary.

The bacteriological findings in acute conjunctivitis are not constant. Thus, in the most violent inflammations gonococcus, diphtheria bacillus, and streptococcus are present; in croupous coating, all bacteria; in trachoma, many of them, but only as a secondary infection.

The majority of conjunctival inflammations are caused by ectogenous infection. They may be due to germs from without, as from dust, finely-atomized droplets, flies, etc. They may also spring from catarrhal affections of the nose and the nasopharyngeal space, or indirectly through the transition of acute, subacute, and chronic, as well as intermittent and exacerbating affections of the lids and lachrymal sac (blepharitis, dacryocystitis). Uncleanliness and unfavorable hygienic conditions promote infections. Personal predisposition and dietetic irregularities also have an influence, as well as temperature, climate, and the seasons.

In Egypt, for instance, conjunctivitis due to Koch-Weeks's bacillus is latent in winter but assumes an epidemic character in summer.

Endogenous toxic and exanthematous hyperæmia of the conjunctiva are accompanied by considerable photophobia and blepharospasm, and under certain circumstances by cedema of the lid and erythema. The two latter, for instance, occur so often in influenza, measles, rubella, and scarlet fever that their presence in an epidemic of these diseases is important for diagnosis. In the early stages of plague, hyperæmia of the bulbar conjunctiva is nearly always present, while conjunctival or episcleral hemorrhages are sometimes seen. There is no increased secretion in spite of intense injection of the conjunctiva, nor do patients complain much of burning in the eyes or of photophobia. Conjunctival hyperæmia in measles usually occurs at the end of the incubation period.

A considerable rise in temperature and serious impairment of the general condition cause diminution of the lachrymal secretion, and this dryness, as well as the reduced palpebral movements, increases the mechanical irritation of the conjunctiva from dust particles present in the air. This leads to continuous lesions of the conjunctival epithelium, which, in their turn, favor the development in the conjunctival sac of more or less injurious germs of all kinds.

The habit of burying the head in the pillows, which is very common with children, likewise plays a part; so does the habit of rubbing the eyes in order to obtain momentary relief from the subjective discomforts—

PLATE IV.

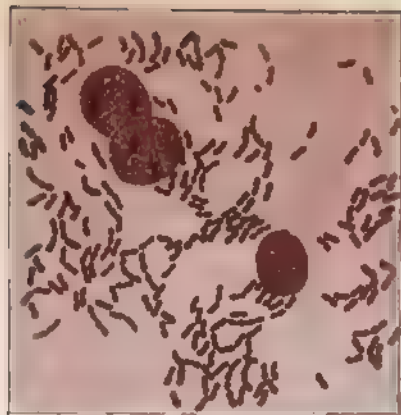


FIG. 1. Diplobacilli (Morax-Axenfeld).

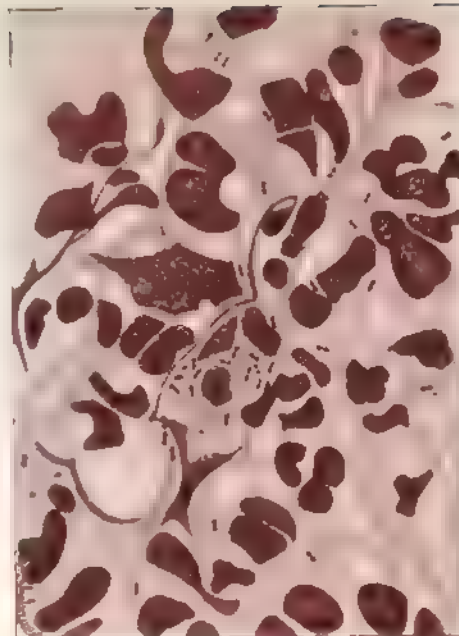


FIG. 2. Pneumococci.

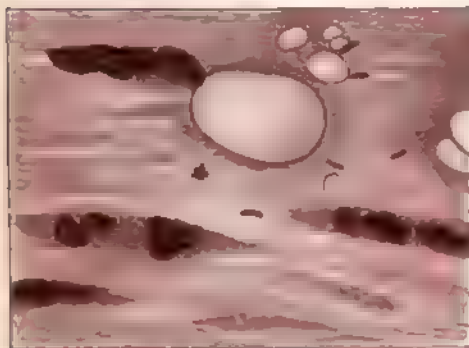


FIG. 3. Xerosis bacilli

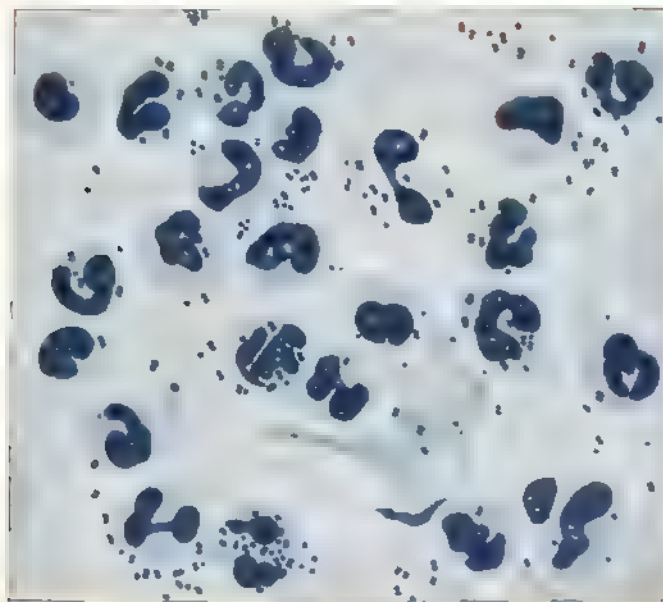


FIG. 4. Gonococci.

photophobia, itching, and burning. Therefore, the younger the patient, the more violent and stormy the course which conjunctival catarrhs generally take. Children are generally more predisposed to such attacks than adults. Both the subjective and objective manifestations are confined within narrower limits in typhoid and rubella, while they are more pronounced in scarlet fever, typhus fever, influenza, and measles.

The following differential points are noteworthy:

In Weil's disease the icteric discoloration of the conjunctiva and sclera is not distinctly visible until a late stage has been reached.

In influenza the conjunctiva may be the only and primary sign of the affection.

In typhus fever there is an injection of the conjunctiva, called "ferret eye," which, together with turgescence of the face and a generally unstable conduct, imparts an appearance resembling intoxication.

In dengue and cholera, hyperæmia and catarrh of the conjunctiva do not set in until the stage of reaction or after. Owing to scarcity of tissue fluid and absence of palpebral movements in cholera, there is early xerosis of the conjunctiva and cornea in the palpebral section.

The so-called plague conjunctivitis, which is usually a primary affection, resembles conjunctival blennorrhœa, except that there is less secretion; but it is aqueopurulent and sometimes brownish, owing to blood extravasation into the conjunctival sac. The pre-auricular and submaxillary glands and the parotid are usually attacked soon after the onset of conjunctivitis.

In cerebrospinal meningitis, at the climax of the disease, the conjunctiva is very often decidedly injected and slightly swollen. Considerable bulbar chemosis, by which the cornea is very often circumvallated, points to suppurative infiltration of the orbital tissue or to suppurative iridochoroiditis.

In conjunctivitis, occasioned by so-called hay fever, catarrhal secretion is usually slight.

In scrofulosis there is a tendency to phlyctenular affections of the conjunctiva and cornea, which appear both during and after the general disease, and are often associated with eczema of the lids or seborrhœa of the palpebral border. This usually occurs in children of weak constitution.

In subacute glanders, measles, and smallpox, blennorrhœiform and diphtheroid conjunctivitis have been observed in the wake of catarrhal conjunctivitis; also facial eczema and extensive excoriations of the lids, with a diphtherial coating.

Coryza in hay fever and the conjunctival irritation during the grass-blooming period are usually moderate in degree, the principal complaints being violent itching and lachrymation.

In difficult dentition of scrofulous children there are sometimes



redness of the eyes and lachrymation, chemosis and photophobia with nictation, blepharospasm of varying degrees, and phlyctenular eruptions of the bulbar conjunctiva. They are immediate consequences of a catarrhal swelling of the nasal mucosa, which occurs oftenest in connection with the eruption of the upper bicuspid.

Unilateral blennorrhœiform conjunctivitis has been observed in stomatitis, and is probably due to an ectogenous infection, for whose growth the reflex irritation of the conjunctiva produces a favorable ground.

Excoriations of the lid are prone to occur when an exanthem involves the lids prominently, too. The conjunctival catarrh, which is usually mucopurulent, is often accompanied by epithelial desquamations and falciform catarrhal ulcers at the border of the cornea.

Diagnosis.—Exaggerated plethora of the vessels of the palpebral conjunctiva, the transitional folds, the caruncle, the plica semilunaris, and the superficial vessels of the bulbar conjunctiva, as well as the diminished visibility of the tarsus and the ducts of the meibomian glands, are strong indications for hyperæmia of the conjunctiva.

Upon development into catarrh there is swelling of the lid. Hyperæmia increases, notably at the transitional folds; lachrymal secretion is increased; the catarrhal secretion, which at first is of an aqueous character, gradually becomes mucous. The lids are agglutinated overnight, notably in the nasal canthus, and there is a desiccated, crusty deposit at the cilia in the morning. The palpebral and bulbar conjunctivæ are often œdematous, and the tissue of the palpebral conjunctiva is loose and puffy. Both subjective and objective signs are usually more pronounced in the evening.

These irritative conditions disappear sooner or later, either gradually or in crises, photophobia being usually the last symptom to disappear.

Chronic conjunctivitis, although its manifestations are like those of acute catarrh, does not often develop from it. If it does it is generally very much less pronounced, but if it continues for a long time, the conjunctiva will be thickened and assume a velvety appearance.

When the catarrh develops into blennorrhœiform conjunctivitis, there is abundant and somewhat purulent secretion. All parts of the conjunctiva are more hyperæmic, and the conjunctiva of the lids and transitional folds are more infiltrated, causing its surface to become uneven. There are often ecchymoses of the conjunctiva, and the lids are more or less œdematous, causing the fissure to appear smaller. The excoriations and eczematous changes of the lid are therefore more strongly pronounced, owing to the continuous moistening of the skin.

Treatment.—Attention should first be paid to any affections of the lid, above all to sycosis of the palpebral border. The conjunctival irritation from coryza in hay fever is most successfully relieved by an im-

mediate change of climate. The most efficacious local remedy in my experience in anæsthesin.

Conjunctivitis, when associated with measles, demands isolation of the patient.

For the cure of photophobia, the patient should be put in a well-ventilated room with a northern or northwestern exposure. The head of the bed should have the windows behind it, as the room will then need no protection from sunlight except for a short period during the day. Bandaging the eye is injurious.

Sensitiveness to comparatively weak light is of nervous origin in conjunctivitis, spring catarrh, keratitis, iritis, chorioiditis, sympathetic ophthalmia, retinitis, cataract, aphakia, etc. Their sequelæ, such as epiphora, pressure in the head, numbness, vertigo, cephalalgia, spots and flashes before the eyes, indistinct vision, myiodesopsia (seeing mosquitoes), and protracted after-images upon looking into artificial light, have been removed instantly by Ruhlmann by instilling aq. zeozoni, three or four times daily. This is a 0.2—0.5 per cent. solution of the orthoöxide derivative of æsculin, and keeps the invisible ultraviolet rays from the eye, rendering protective spectacles, veils, screens, etc., permanently unnecessary. It has also a favorable effect upon spastic contraction of the eyes, light conjunctivitis, blepharoconjunctivitis, and chronic blepharitis.

This is especially true of conjunctivitis in measles, so much so that with instillation of zeozone water Ruhlmann allowed even the initial eruptive fever to run its course in a light room. Grave iritis also heals rapidly in a light room without protective spectacles if this instillation is used. The solution has a light yellow to brown color and is not easily washed off by the lachrymal secretion; it stains the skin light yellow, but not the cornea or conjunctiva. The yellowish discoloration will disappear upon touching it with diluted acetic acid.

High degrees of photophobia and the inclination to rub the eyes are alleviated by cocaine instillations (cocain. salicyl. 0.025, aq. dest. 5.0, twice to three times daily). If rubbing, nevertheless, continues, the elbow-joints are placed in cardboard splints. To relieve itching and burning, cooling compresses dampened with a 3 per cent. boric acid solution are applied to the eye for five or ten minutes three times daily. Von Reuss treats the very annoying complaints in acute catarrh of the conjunctiva with the faradic current, once or several times a day, as indicated.

If there is much secretion of the conjunctiva, the lids are wiped off at frequent intervals with sterilized cotton tips, and some astringent is instilled repeatedly. Sodium bicarbon. 0.1–0.2 : 10.0 aq. dest. is the weakest; among the stronger solutions I prefer silver nitrate 0.01–

0.02 : 5.0, once every morning. The preparation should be often made fresh from a stronger solution which has been preserved in a dark glass.

Instillation of any kind of drops into the eye requires the greatest caution, as impure material may cause a dangerous infection, if, for instance, the corneal surface is even abraded. Every solution used for instillation must, therefore, be sterilized. Admixtures are sometimes harmful; sublimate, for instance, even when considerably diluted, is liable to injure the conjunctiva. The solution may be changed by forming deposits, as, for instance, in the case of pilocarpine mur.

The solution, after having been sterilized by boiling, must be kept germ-free. It should, therefore, be boiled again and again before using it in continued applications, or in operations on the eye, of whatever nature. This no doubt impairs the efficacy of physostigmine and cocaine solutions. In others, the concentration increases by evaporation and may finally exceed the admissible percentage. The practitioner should, therefore, prepare an alcoholic mother solution of the most frequently used remedies (atropine, cocaine, etc.), from which smaller quantities in 5-10 Gm. solutions may be prepared as needed.

Many physicians use pipettes, which, with their contents, can be sterilized in live steam. The essential point is to see that they are not infected during use, and do not come in contact with the palpebral border, cilia, or conjunctiva. After use they should be so replaced in the container that their point does not touch the mouth or the inner wall of the glass. It is advisable to heat the solution before instillation to 25-30° C. (77-86° F.), not only because a warm drop is more rapidly absorbed by the conjunctival tissue than a cold one, but also because there is no reflex closure of the lids and struggling. This applies particularly to atropine, physostigmine, and pilocarpine, where an immediate and lasting effect upon the pupil is desirable.

In case of need, a clean glass rod, rounded at both ends, such as is used in applying ointments to the eye, may be used for instillation (see below). These sterilized glass rods should be preserved in a concentrated salt solution in a cool place, the solution being well covered and renewed every few days. Before use, the salt solution attaching to the rod is removed by flooding the container with pure, fresh water. The fluid, or ointment, attaching to the glass rod after use is carefully wiped off with sterile gauze. It is advisable to give parents written directions, in plain words, as to the use of the solution or ointment, with a caution to read the directions each time before application.

The procedure should be explained to the patient, if possible, so that he will not pinch his lids together, but keep the eyes gently closed for a few minutes after the instillation.

After the instillation of cocaine, the lids are covered for five or ten

minutes with gauze, moistened with a 3 per cent. boric acid solution, so as to prevent the drying of the cornea. The instillation is made as follows (Fig. 24): The upper lid is slightly raised. The patient turns the sound eye away from the affected eye, if possible, so that he looks to the left when the instillation is to be made in the right eye. A drop is then allowed to fall slowly upon the scleral cornea from upward and outward, after which the eyes are closed.

The physician should always personally demonstrate how instillations should be made. Cocaine and atropine should be used sparingly.

FIG. 24.



The instillation of drops.

If combined with each other, they may cause pus-like corneal infiltration, which may be mistaken for a septic process and cauterized, to no purpose whatever.

Ointments may be used instead of an instillation when a prolonged effect is desired or the treatment is protracted. The ointment should, of course, be well prepared and stable, and carefully preserved in a dark, well-closed jar. An amount about the size of a small pea is applied with a clean glass rod, rounded at both ends. The lower lid is lifted away and the ointment applied from the external canthus. The patient lightly closes his lids, and keeps them gently closed while the rod is being withdrawn. Delicate rubbing of the eye with a small, roundish, absorbent cotton pledget will distribute the ointment evenly in the conjunctival sac. The eyes should remain closed for a few minutes. As ointment base amyloglycerin, lanolin, and vaseline are used. American vaseline (Chesebrough) is considered particularly stable.

It is not advisable to use silver nitrate in the form of an ointment, on account of the possible presence of argyrosis. Oily collyria are also adapted to this use. In Germany the eye drops manufactured after v. Pflugk's directions have met with particular favor (1 per cent. acoin, 1 per cent. atropine, 1 per cent. cocaine, 1 per cent. pilocarpine, $\frac{1}{2}$ and 1 per cent. physostigmine, $\frac{1}{4}$ per cent. scopolamine).

Silver nitrate and physostigmine solutions should always be kept in a dark glass, owing to the danger of decomposition. Physostigmine solution is more stable if one or two drops of sulphurous acid are added to 10 Gm. of solution.

To prevent agglutination of the lids, an ointment of white precipitate (0.05 to 0.1; vaseline, lanolin $\bar{a}\bar{a}$ 5.0) is applied with a glass rod to the borders of both lids before retiring.

The widely-used cold compresses in high degrees of blennorrhœiform conjunctivitis are objectionable, because they are apt to increase the maceration due to the conjunctival secretion, as well as the irritation and swelling of the conjunctiva. A better method is to keep the palpebral border scrupulously clean, and to irrigate the conjunctival sac gently several times with a tepid 3 per cent. boric acid solution, kept in a suitable container.

In suppurative conjunctivitis, silver nitrate will effect the most rapid cure. The lids are everted, leaving the cornea covered. The exposed palpebra conjunctivæ and the transitional folds are delicately painted several times with a soft brush dipped into a freshly-prepared 1-2 per cent. solution of silver nitrate—first the upper, and then the lower lid—producing a superficial scab formation of light blue appearance. The surplus quantity is rapidly removed with a second brush, which has been dipped into a 1-2 per cent. salt solution or boiled, tepid water.

If there is any swelling or blepharospasm, the eversion of the upper lid may be difficult. The following method is the best: Place a lachrymal sound, a thin round pencil or similar object upon the upper lid where the transverse fold appears, and at the point which corresponds to the upper border of the cartilage. Do not displace the skin of the lid, nor press upon the eye. With the disengaged hand draw the border of the lid downward and slightly forward by the cilia, and turn it over the sound against the supra-orbital margin, where it can be easily kept with the finger-tips by just holding or pressing it against the margin. The procedure is facilitated by letting the patient look downward upon his outstretched hand, and also by previous instillation of a 3 or 4 per cent. cocaine solution. In case of need, a slight dash of anæsthesia with ethyl bromide or Billroth's mixture may be resorted to without apprehension.

The burning and irritation which follow the procedure will subside

within a few hours, giving way to a feeling of relief. The scab desquamates at the same time. Cauterization should not be repeated, unless the secretion, which varies according to the stage of the catarrh, again increases, and then only after the first scab has disappeared. As a rule, one application is sufficient, and it is best to make this in the early part of the day.

Marginal ulcers of the cornea, which may complicate the affection, do not contra-indicate painting.

After the purulent secretion has greatly diminished, astringent remedies are sufficient for light catarrhs. Those most in use are lead acetate in $\frac{1}{5}$ - $\frac{1}{4}$, cupr. alum. in $\frac{1}{10}$ - $\frac{1}{5}$, zinc sulph. in $\frac{1}{3}$ - $\frac{1}{2}$, zinc salicyl. in $\frac{1}{4}$ - $\frac{1}{2}$ per cent. solutions. They are applied alternately once or twice a day, in the morning.

The conjunctivitis of the Koch-Weeks's bacillus may occur as an acute epidemic, and is highly contagious. The pneumococcus conjunctivitis is contagious under certain conditions. Both may require the exclusion of the patient from school attendance, or even the closing of schools, and similar measures.

The conjunctivitis of the Koch-Weeks's bacillus is particularly prevalent during the summer. The leading symptom in pneumococcus conjunctivitis, which is also very prone to attack children, is considerable involvement of the bulbar conjunctiva with hæmorrhagia, even in lighter cases, and the sudden cessation of inflammatory manifestations.

Chronic catarrhal conjunctivitis (Plate V, Fig. 1), also called *catarrhus siccus*, is a highly contagious disease and, consequently, is often found endemic or affecting an entire family. The diplobacillus Morax-Axenfeld has very often been found bacteriologically (Plate IV, Fig. 1). There are burning and itching of the eyes, and a sensation as if they contained fine sand, or the lids may feel dry and heavy, especially on awakening. These manifestations vary according to the sensitiveness of the eyes, particularly of the palpebral skin, to chemico-traumatic irritation, sharp wind, etc. Sometimes they are so slight as to escape the patient's observation. Artificial light, hot or dusty rooms, and accommodative efforts of the eyes exacerbate the complaint. Defective nutrition, constipation, chronic nasal complaints, acne, and chronic eczema of the face serve to keep up the conjunctival irritation. This condition may also be produced by disturbances in the lachrymal secretion, blepharitis and positional anomalies of the lids; by molluscum contagiosum or small papillomatous eminences at the intermarginal fringe of the lids, and stagnation of secretion, or concretions in the meibomian glands.

It is important to distinguish this conjunctivitis from the so-called *blepharitis angularis*, an erythematous reddening of the cutis in the

palpebral commissures, and the weeping appearance of the eyes. Small, light follicles in the transitional folds, especially the lower one, have been observed as concomitant manifestations, and marginal ulcers of the cornea as a sequel. The obstinacy of some cases has been explained on the assumption that the diplobacillus is able to persist in the glands of Krause for a long time.

Treatment.—Dietetic and hygienic prophylactic measures which accord with the etiology should be instituted, proper spectacles prescribed for protection or correction, and neighboring pathological processes removed. Where the secretion contains the diplobacillus, instillations, twice or three times daily, of 1/5–1/2 per cent. zinc suphate have done good service, provided they are continued for a sufficiently long time, from two weeks to two months after the affection has apparently run its course. According to Knapp, 2 per cent. resorcin, instilled twice or three times daily, gives less cause for complaint than 1/2 per cent. zinc. The following ointments are serviceable for application to the lids overnight: v. Sehlen-Peters zinc ichthyol paste; emplastr. lithargyr. spl. (free from glycer.) 5.0; ungt. paraffin. (paraff. liquid. āā 2.5; aq. dest. 1.0; rec. parat.; ad o. opac. claus.); Sattler's zinc sulphate ointment (zinc. sulph. 0.02; ichthyol 1.0; white vaseline 10.0). In obstinate cases, intermittent gentle massage of the conjunctiva may be tried, with instillations of collyr. adstr. lut. (1.0 : 3.0 aq. dest.), suprarenin boric. with novocaine and sodium biborac. (sodium biborac. 0.2; novocaine 0.025; suprarenin boric. 1.0 : 1000.0, gtt. iii, ad. 0.9 per cent. sodium chloride sterilis. 10.0. MD ad. vitr. opt. claus.). Immersion in water at room temperature, in the morning and evening, alternately closing and opening the eyes to a slight extent, often procures a pleasant sensation.

Persistent conjunctival catarrh has been successfully treated by Luciani and Peters with ichthyo-zinc ointment. Luciani recommends in addition irrigations with a 1–10 per cent. solution of ichthyol. ammon.

Infiltrations and ulcers of the cornea may require clinical treatment. (For further particulars see chapter on "Cornea.")

2. ECZEMATOUS CONJUNCTIVITIS AND KERATITIS

(SYN. C. PHLYCTENULORO-PUSTULOSA, IMPETIGINOSA, LYMPHATICA, SCROFULOUS OPTHALMIA.) (FIG. 25.)

This disease deserves particular attention on account of its prevalence. It is very prone to reoccur, and, while usually of mild course, it is sometimes accompanied by chemosis and follicular or catarrhal conjunctivitis. It is attributed to endogenous toxins of tubercle bacilli, and to an infection of *Staphylococcus aureus*. The clinical picture is as follows: Subepithelial collections of round cells, "phlyctenules" accompanied with pericorneal and subconjunctival injection, occur in

PLATE V.



FIG. 1. Blepharoconjunctivitis (Morax-Axenfeld)



FIG. 2. Phlyctenular conjunctivitis.



FIG. 3. Crupous conjunctivitis



FIG. 4. Blepharitis neonatorum.



FIG. 5. Follicularis.

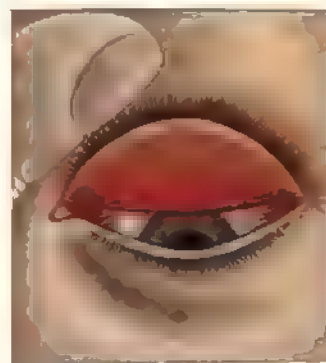


FIG. 6. Fresh trachoma

various sizes and numbers in the bulbar conjunctiva and the cornea. The favorite location of these small nodules or phlyctenules is at the junction of the cornea and sclera (Plate V, Fig. 2). They disintegrate into roundish superficial ulcers which often heal in a week or two, leaving no visible traces. In the more obstinate forms, however, the cellular infiltration spreads to the deep structures, leading to local tissue degeneration of the conjunctiva or cornea. When corneal infiltration develops by superficial extension, it is spoken of as "vascular ribbon," "bundle-shaped keratitis," "keratitis fascicularis," or "herpetetic bridge" (Plate VI, Fig. 6). The phlyctenulae may appear first in the cornea and lead to extensive ulcers of that membrane. Corneal opacities with consequent reduction of vision depend upon the area of the cornea involved and the

depth of the ulcers. Lowered vision from these causes is a frequent occurrence. Phlyctenules of the palpebral conjunctiva, which are very rare, are usually situated in the vicinity of, or upon, the intermarginal border.

Subjective symptoms of irritation consist in a sensation of pressure in the eye, photophobia, lachrymation, and blepharospasm. They are often slight or absent, especially when the phlyctenules of the bulbar conjunctiva are isolated and are not near the limbus. They are of a moderate character when the disease is limited to



Habitus scrofulosus.

multiple small vesicles situated in the limbus. As a rule, they are very pronounced in miliary conjunctivo-keratitis and in progressive vascularized infiltrates (fascicular keratitis, etc.).

Conjunctival phlyctenules of the corneal margin are of light yellow or reddish color, due to circumscribed injection of the vessels in that region, notably the subconjunctival, and also those of the pericornea and posterior conjunctiva. Phlyctenules located in the zone of muscle insertions, which are often attended with swelling of the bulbar conjunctiva in the region of the lid space and acute catarrhal conjunctivitis, have a dirty gray appearance.

If the latter are numerous and grow closely together, they coalesce into a broad area of infiltration, whose epithelial covering undergoes early desquamation (ulcus elevatum). If this area advances toward the corneal margin, it may give rise to a deep marginal corneal infiltrate.

I have several times observed the progression of a conjunctival pustule into the deep parts toward the sclera, so that, after cleansing, the ciliary body was visible. A blue-gray scar remained.

In miliary phlyctenular conjunctivitis numerous minute epithelial eminences appear with considerable pericorneal injection, resembling fine sand-like granules. They develop very rapidly in the circumference of the limbus and in the cornea, sometimes more numerous in one than in the other. Confluence of adjacent phlyctenules may lead to corneal ulcers with many new capillaries leading to abscess-like infiltrates and deep scars. They are either superficial or deep, the latter especially if they occur near the centre of the cornea.

Pustular conjunctivitis (*phlyctænulosa maligna*) is much more dangerous, and nearly always accompanies catarrhal conjunctivitis. The eminences are at first whitish-gray, changing later to light yellow, have a diameter of 3 to 4 mm., and are almost exclusively located in the lower half of the corneal margin. A deep, crater-like defect, with perforation and marked cloudiness of the surrounding corneal tissue, develops very rapidly. Prolapse of the iris (Plate VII, Fig. 1) and subsequent leucoma may occur (Plate VI, Fig. 3), partial staphyloma, two pustules coalesce and the corneal margin is somewhat extensively destroyed.

Phlyctenular conjunctivitis attacks preëminently children of the "scrofulo-lymphatic" type.

Conjunctival catarrh, acute exanthem, and skin eruptions like eczema, intertrigo, prurigo, urticaria, the various forms of blepharitis, exanthematous eruptions of the face, pediculosis capitis, and uncleanness, are of etiological importance.

The cause of phlyctenules in non-tuberculous children is assumed to be "lymphatism," "oxypathy," or "exudative diathesis," due to overnutrition with fat-depositing food, and a resulting auto-intoxication on the part of the intestine. Consequently, owing to hypersensitiveness to bacterial proteins, the conjunctival and corneal tissues of these children are readily infected, especially if their power of resistance has been impaired by a previously-existing tuberculous infection.

As a matter of fact, in *acne rosacea*, in which auto-intoxication is a large factor, efflorescences occur at the limbus of the bulbar conjunctiva which cannot be distinguished, histologically, from exanthematous conjunctivitis.

Colombo found indican, and in other cases urosein, in the urine of patients suffering from keratoconjunctivitis, showing that there was abnormal fermentation in the intestine, and auto-intoxication.

The affection terminates in most cases with the disappearance of these constitutional tendencies, which is to say with the advent of

PLATE VI.



FIG. 1. Circumferential trachoma.

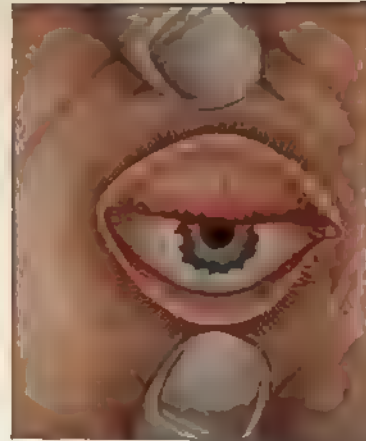


FIG. 2. Spring catarrh.



FIG. 3. Leucoma corneae adherens.
Sequel of Fig. 1, Pl. XVIII

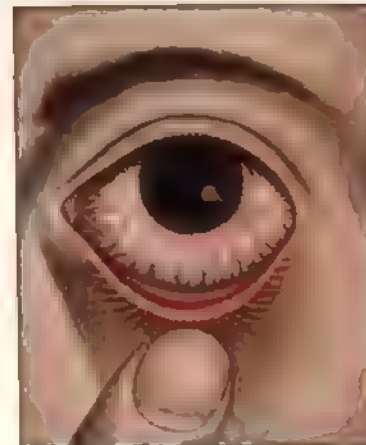


FIG. 4. Corneal ulcer. Corneal macula.

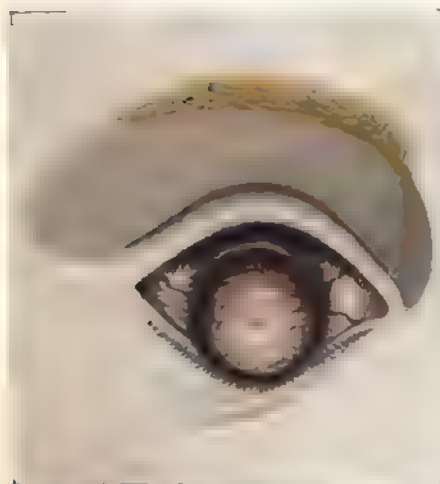


FIG. 5. Total staphyloma.

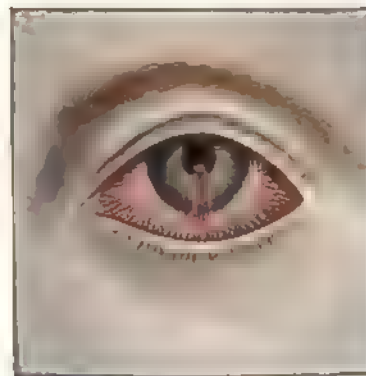


FIG. 6. Keratitis fascicularis.

The *prognosis* in the simple and miliary type of phlyctenular conjunctivitis is favorable; it is also good in "elevated ulcer," which is confined to the conjunctiva. The prognosis of phlyctenular corneal affections is less favorable, and that of the pustulous form very serious, as neglected cases are not rare in which the corneal process in one eye has already taken a serious turn. The general prognosis is doubtful, or favorable, only under good conditions.

There is a tendency to relapse, due to local results of the affection, such as rhagades of the external canthus, blepharophimosis and ectropion. They are also due to the general condition of these children, who almost exclusively belong to the poorer classes. Their vitality has often been impaired by antecedent infectious diseases, by carious processes of the bones, notably the petrous bone (otorrhœa), and by eczematous eruptions.

Adenoids, enlarged tonsils, pharyngitis, hyperplasia of the mucosa of the velum, uvula and arches, and more or less infiltration of the deep lymph-glands, are contributory factors. Many cases present chronic, mucous, purulent catarrhs of the nose and respiratory tract, infiltration and eczema of the nostrils and upper lip, or erosions of the septum which often cause epistaxis. Swelling of the septum, and polypus or papillary hyperplasia of the turbinates, are not rare occurrences. If these conditions have existed for a long time, recurrent erysipelas is common, leading to thickening and sclerosis of the alæ nasi, lips, and cheeks, and sometimes of the lids.

Von Hoffmann was the first to point out the connection of eczematous phlyctenular conjunctivitis and keratitis, obstinate eczema and inflammation of the lid margins, with the affections of the lachrymal ducts and the tonsillar affections and concretions, as well as the value of nasal and facial treatment to scrofulous eye patients. At the Juvenile Station of the Ophthalmological Clinic of the Munich University all scrofulous children are examined once a week by a rhinologist, and treated according to findings.

Treatment.—The favorable prophylactic influence of timely hygienic and dietetic measures is demonstrably better in this than in any other affection of the eye. For this reason, hospital treatment with a conscientious nurse effects improvement in a very short time, even in neglected cases. Unfortunately, at home the injunctions are only imperfectly obeyed, if at all, so that the disease breaks out afresh. This state of things can be averted to a certain extent by having the nurse instruct the mother in the care of the child on its discharge from the hospital.

Printed instructions in plain language, containing the most important principles of nutrition and general hygiene, should be given the

mother. They should point out the value of hardening children against catching cold by being in the fresh air, the value of correct respiration, and give instructions for the care of the eye.

Different bathing utensils and water should be used for the head and body, so that the eczematous products may not come in contact with the healthy parts of the body.

The first baths are given at 33° C. (91.4° F.), twice or three times a week, lasting from ten to fifteen minutes. Then a jug of water about four degrees colder (84.2° F.) is poured over the body—not over the head—followed by rubbing with a dry friction towel. This is best done before the evening meal, so that the child may be put to bed immediately. The temperature of the bath is gradually lowered to 26° C. (78.8° F.), and the temperature of the water to be poured over the body is also gradually lowered by one degree down to 20° C. (68° F.). In very delicate and susceptible children the procedure should begin with dry friction, after which the various parts of the body are washed singly and covered one after the other. But these instructions are often not followed long enough. In most cases hardening sufficient to render children immune to colds is achieved only after one or two years.

Salt or brine baths, twice or three times weekly, are also very useful; if there should be any important skin eruptions, chamomile or bran baths should be given.

The windows of the bed-room should be open day and night, even in wet weather and in winter. Draught near the bed is to be avoided.

The clothing should be loose, so that respiration, digestion, and motility are not interfered with. Collars should be wide, as narrow ones impair the return flow of the blood from the head. Neither boys nor girls should have their clothing tightly belted in. Narrow garters should be forbidden; thick headgear is injurious.

Clothing which is too warm relaxes the vessels of the skin and thus conduces to catarrh of the upper respiratory tracts and, consequently, a return of the ocular affection. Cold hands and feet must be avoided.

The eyes should be kept clean, with a strict injunction not to wipe them. Cleanliness of the oral cavity is likewise of great importance. Children brought up on cow's milk and artificial preparations often suffer from stomach-ache, aphthæ, and caries of the milk teeth, often leading to acute and subacute infiltrations of the submaxillary glands. The best protection is mechanical cleansing of the teeth, especially the interstices, and cleanliness of the oral cavity by gargling with water or a warm $\frac{1}{2}$ to 1 per cent. salt solution morning and night and after every meal. The gargle should be forcibly thrown about in the mouth. Broken teeth or roots which cannot be preserved should be removed; every cavity, even the smallest, should be filled, even in the first set.

PLATE VII.



FIG. 1 Prolapse of iris after perforation of a phlyctenular marginal ulcer

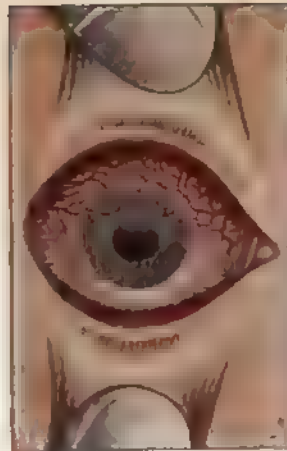


FIG. 2 Pannus keratitis.

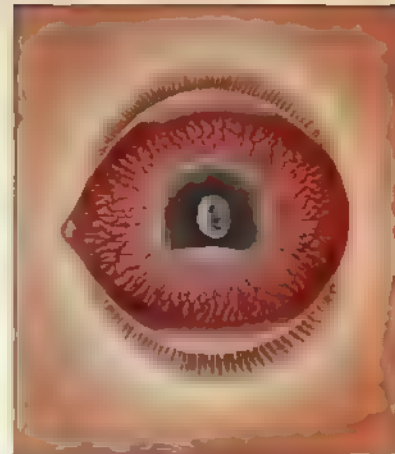


FIG. 3 Serpiginous ulcer of the cornea with hypopyon



FIG. 4 Keratitis disciformis post-vaccinosa.

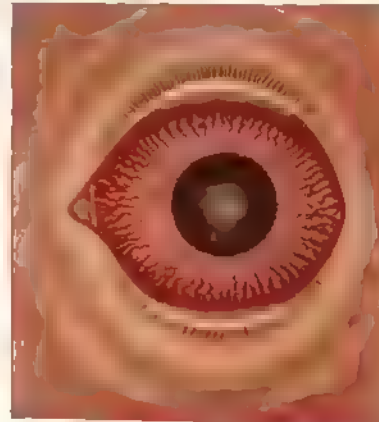


FIG. 5 Parenchymatous keratitis.



FIG. 6 Avascular parenchymatous keratitis



FIG. 7 Deep vessels in parenchymatous keratitis.

Insufflation of boric acid, soziodol salts, and general dietetic strengthening measures are indicated in light hyperplasia of the lymphatic faucial ring.

For recurrent or habitual chronic tonsillitis, v. Hoffmann and Schmidt advise incision of the tonsils. Lénart recommends *morcellement* in the following manner:

The hyperplastic tonsil is first ablated, after which every diseased part is removed with Hoffmann's double curette, with the guide of the sound, until the sinus tonsillaris becomes visible as a small spot. In order to remove thoroughly all the affected parts, it may be necessary to hook the anterior arch of the palate and cleanse the region below; this may require incision of the arch. Hemorrhage is less than in tonsillotomy, as is also the after-bleeding on the second or third day of the operation. The proceeding is not very painful, and in from eight to twelve days patients are fully restored.

The removal of adenoid vegetations and enlarged tonsils often effects an immediate, extensive, and permanent improvement, so much so that eyes which even in the intervals between exacerbations are always red, from weeping and photophobia, suddenly become free from irritation and assume a normal hue.

Of equal importance is the removal of obstacles to nasal respiration, such as exostoses of the septum, polyps, narrow posterior nares, chronic coryza, etc.; so is the treatment of excoriations and ulcers, which in atrophic rhinitis usually occur at the nasal septum and the locus Kieselbachii, as the result of scratching the nostrils. Habitual epistaxis may result when the vessels of the mucosa are very fragile, and the loss of large quantities of blood may produce permanent debility of the constitution. The nose is cleansed by snuffing up a tepid 3 per cent. boric acid solution several times every day, or by drenching it with the nasal bath as recommended by Fränkel and Moritz Schmidt. Insufflations of soziodol and nosophen, with talc. venet. (1 : 2-3), are made with a blower or a rubber tube of 3-4 mm. diameter attached to a glass headpiece bent at a right angle. Another method is to introduce a sodium soziodol solution in spoonfuls or to aspirate it, inserting alternately into each nostril a cotton pledget coated with yellow precipitated ointment.

Exclusive feeding on sterilized or so-called fat-milk, which has been widely used in the last ten or twenty years, is injurious. Nurslings should be gradually accustomed, as early as the seventh week, to the ingestion of lacto-vegetable diet containing nutritive salts in the shape of orange and other fruit juice. From the sixth month, mashed vegetables are given, first spinach and carrots, carrot soup, then a little meat juice and any other vegetables which are taken without repugnance.

Older children with manifest exudative diathesis should be guarded against overnutrition from a diet overcharged with fat, sugar, eggs, butter, and meat juice, since this invites decomposition. The milk ration is reduced to $\frac{1}{2}$ – $\frac{1}{4}$ litre ($1\frac{1}{2}$ pint) a day, while fruit, fruit juice, and vegetables should be given freely. In some cases the diet should be purely vegetable.

Proper mastication and salivation of the food are of great importance. Stomatitis, if present, should be removed by cold food and washing out the mouth with a $\frac{1}{2}$ –1 per cent. solution of hydrogen peroxide or tincture of krameria.

It is most important to intestinal fermentation and abnormal decomposition, as well as to relieve constipation. It is equally important to arrest diarrhoea. Pulvis antiscrofulosus, as recommended by v. Graefe, is composed as follows: Hydrarg. chlorat., antimonium chlorate, pulv. fol. conii mac. $\bar{a}\bar{a}$ 0.06, sacch. alb. 0.5; M. F. pulv. D. tal. dos. No. xii. S., one powder two to four times daily. Von Hoffmann recommends ichthyol (ammon. sulfo-ichthyolic.; aqu. dest. $\bar{a}\bar{a}$ 10.0, 3–10 drops three times daily; children to receive one drop more for each year of age). M. Schmidt, H. Schulz, and I have found the following very efficacious: Flor. sulf. and sacch. lact. $\bar{a}\bar{a}$, $\frac{1}{2}$ to $1\frac{1}{2}$ teaspoonfuls, well stirred in half a cup of hot milk, and taken before meals so as to procure a normal stool. After four to six weeks, the medication is interrupted for a month; it is then again taken for a month, and so on for six to twelve months.

Other medicaments are: Cod-liver oil, ferrum iodide, ferratin iodide, ferratose iodide, arsenious ferratose, ferrum iodide and cod-liver oil, Duerkheim mineral water (almost free from iron and containing much arsenic), Guben water, Levico water, Roncegno water; and, if there are distinct manifestations of pulmonary tuberculosis: guaiacol. carbon. 3.0, ol. morrhue. 200.0; one tablespoonful twice daily.

Further experience is needed in the use of tuberculin in this condition. Bernheimer and others found it very successful, while Koster sounds a warning against it, if there are extensive tuberculous foci at other parts of the body.

In moist facial eczema, which often invites a recrudescence of the affection, the parts should be dusted with a powder composed of equal parts of starch and boric acid, or powders containing zinc or nosophen. In superficial eczema scabs should be macerated by the application of boric ointment spread on lint, followed by painting with a 1–2 per cent. solution of silver nitrate. Rhagades of the nostrils and upper lip should be carefully touched with a finely-pointed, smooth mitigated stick. This usually requires light anæsthesia. Scabs and crusts caused by cauterization are covered with boric or zinc ointment, and will fall off

spontaneously. Soon afterward zinc-nosophen paste (nosophen 0.5, zinc. oxydat. amyl. pur. āā 1.0, vaselin. alb. ad. 10.0) and myrtillin (Winternitz) should be applied. If ointments are not tolerated, as is the case with a tender skin, the scabs are removed with 3 per cent. boric water, after which the spots are powdered with pure starch or equal parts of starch and zinc oxide āā.

Ulcers of the skin are bandaged with iodoform gauze, immersed in aluminum acetate.

Pediculosis capitis is removed with sabadillic vinegar or vinegar and liquor van Swieten āā, provided the scalp is intact; in impetigo equal parts of petroleum and olive oil are applied. The scratches heal after six to seven days, after which the head is washed with water and black soap and with 0.5 sublimate in two pints of vinegar.

The removal of neighboring glandular infiltrations or otorrhœa often effects a permanent cure of the ocular complaint.

Local Treatment.—If the lids and their conjunctiva are not involved, and in the absence of fresh suppuration or serpiginous infiltration or loss of corneal substance, it will be sufficient, in addition to cleansing the eyes with tepid 3 per cent. boric acid solution, to make daily insufflations of calomel, boric acid, iodoform, or nosophen. This is best done in the forenoon. Or massage may be instituted with yellow ointment (puleolum luteum Schweissinger s. ungt. hydrarg. oxydat. parat. pultif. 0.1 : 10.0).

According to Luciani and Peters, ichthyol zinc paste, applied with a small glass rod to the conjunctiva, is attended with excellent results. Similar results have been reported of Lassar's paste, consisting of zinc oxide and olive oil (40 : 30), and gallicin. The burning sensation caused by the latter remedy is relieved by cold application.

To prevent recurrence, this irritative treatment is continued for two or three months after the disappearance of the phlyctenules. Parents may be safely entrusted with the powdering manipulations, provided they have received careful directions.

Washing out the eyes with cold water and soap, followed by immersion of the face in clean, fresh water, in which the eyes are several times opened and closed, if habitually followed morning and evening, very often prevents relapses.

Calomel should be kept in a tightly-closed jar, on account of its liability to absorb water and become lumpy. It must not be polluted with bichloride of mercury. If iodine is administered internally, there may be conjunctival irritation due to the formation of mercury biniodide and protiodide from the free iodine excreted in the tears. This can be prevented by administering the iodine preparation several hours before or after the calomel powdering.

In powdering the eyes with calomel (hydrargyr. chloride mite vapore paratum), boric acid, iodoform, or nosophen, the finest possible distribution should be effected. This may be done by means of a brush with very fine, silky hairs and the shape of a broad cone (Fig. 26). When using blowers, any undue pressure may work harm, inasmuch as little lumps of calomel may erode the conjunctiva. The lower palpebral conjunctiva is everted, and the adjacent lower fornix is powdered freely, in order to make sure that the powder really comes into contact with the affected parts of the conjunctiva and cornea. Immediately afterward, the eyes should be gently closed for a minute.

Powdering with airol, iodoform, xeroform in infectious ulcers and injuries of the eye, of gallicin and tannic acid-boric acid (1 : 3; exactis-

sime pulveris.) in chronic conjunctivitis and acute granular conjunctivitis, and anæsthesin in hay fever conjunctivitis, requires the same technic.

Photophobia and blepharospasm are relieved by instillation of zeozone water (see p. 131), of paranephrin or suprarenin boric. 1 : 10,000 with 1 per cent. cocain. hydrochlor., or by application of cocaine-suprarenin ointment (cocain. mur., suprarenin 1 : 1000 āā 0.1, vaseline 10). The faradic current is indi-

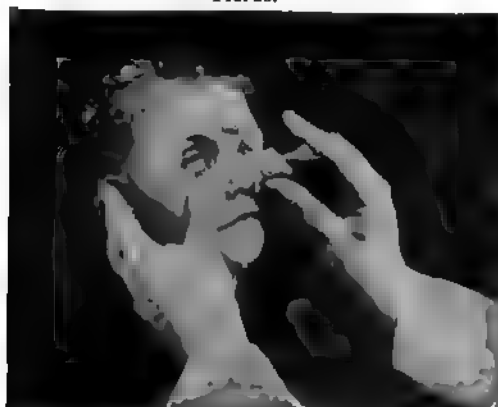


FIG. 26.
The insufflation of powder in the eye.

cated for the cure of photophobia, which sometimes persists after eczematous conjunctivitis without any demonstrable cause.

Mydriatics are indicated only in severe blepharospasm and pronounced pericorneal injection and hyperæmia of the iris, even when the cornea is but slightly involved. Mydriasis is maintained until the bulbar irritation subsides or disappears.

In the absence of hyperæmia of the iris, the sharply circumscribed marginal vesicles and infiltration of the cornea, which occur without any particular injection of the bulbar conjunctiva, are treated with physostigmine (1 per cent.), pilocarpine (2 per cent.), morphine (1/10–1/5 per cent.). These may be used singly or combined, as indicated.

For treatment of the lid affections, compare p. 89. See also "Eczematous Ulcers of the Cornea," p. 198.

Ungt. hydrarg. iodide and biniodide in powder form, manufactured after v. Ammon, contain the active principles in exceedingly fine distribution, surpassing that of the protoxide in Pagenstecher's ointment.

Thus, "the irritation of the eye, which can never be entirely avoided, is reduced to a minimum."

Yellow oxide of mercury ointment, 1 per cent., is recommended for marginal phlyctenules, corneal phlyctenules with slight irritative manifestations, for blepharospasm caused by rhagades of the external lid commissure, and for scrofulous affections of the lid border. "Increase of the inflammatory conditions of the pathological tissues, giving the patient an unpleasant sensation, must be avoided; likewise simultaneous internal use of iodine."

Ointment of biniodide of mercury, 0.3–0.5 per cent., is almost exclusively used in chronic blepharitis with exaggerated scale formation. It is applied to the border of the closed lids and spread with a glass rod, so that the ointment may penetrate well into the roots of the cilia. It is allowed to remain there from three to six minutes with closed eyes, after which the borders of the lids are thoroughly rubbed with a moist gauze sponge, so as to remove the adhering scales. One application may suffice, or several may be required, but the treatment must be continued until the last scales have been removed. If, however, the conjunctiva shows symptoms of irritation, the applications must cease. In that case, or in the presence of marked scab formation, the lids are macerated the previous evening by 1 per cent. yellow oxide of mercury ointment applied on a bandage. This treatment is repeated every four, eight, or twelve days, according to the degree of the affection, and may have to be continued for several months. "It should be applied by the physician himself, because patients cannot be expected to do so correctly, for technical reasons."

It has repeatedly occurred that after vaccination, ever so skilfully performed, children of tuberculous parents were attacked by violent and rapidly developing scrofulous ophthalmia. Although there is no proof of causal connection, it is advisable not to vaccinate children suspected of a tuberculous or scrofulous disposition until the manifestations have sufficiently developed to become apparent to the parents.

3. VARIOLAR PUSTULES OF THE CONJUNCTIVA

These pustules usually occur on the bulbar conjunctiva in close propinquity to the lower corneal border. Becoming confluent, they resemble a yellowish-gray diphtheroid infiltration. They measure about 5 mm. in diameter and advance a short distance toward the corneal margin. But even with considerable extension backward the lower transition fold is never reached. Even after confluence of the flat, moist pustules, the notches toward the inferior fornix indicate the site of the original pustules. This often produces marginal ulcers or deep, suppurative infiltrations. Destruction of the cornea, prolapse and staph-

yloma of the iris, purulent iridochorioiditis, and panophthalmitis are not impossible sequelæ. Therefore daily inspection of the affected eye is imperative.

Pustules originate from implantation of the organisms from the lids upon the globe. Smaller pustules of the bulbar conjunctiva develop from pustules of the palpebral conjunctiva, and result from contact with pustules of the marginal border. They are less menacing, as they are more distant from the border of the cornea.

Treatment.—Owing to the marked swelling of the lid the destruction of the pustules by means of silver chlorate fused to a sound and immediately neutralized is practically impossible. In most cases we have, therefore, to be content with instillations of fresh chlorine water, diluted with distilled water, or of well-filtered lemon juice (equal parts of lemon juice, purest glycerine, and 1/10 per cent. solution of morphine).

4. VACCINOLA AND VARICELLÆ

The irritative manifestations, swelling, injection, and chemosis of the conjunctiva following vaccinola of the palpebral border are more marked if the vaccine pustules develop upon the conjunctiva itself. The cornea, too, is more threatened by ulcers or parenchymatous infiltrates. Vaccine ulcers with swelling of the pre-auricular gland and febrile manifestations, and superficial losses of substance covered with a whitish-gray, easily detachable layer, usually heal promptly without leaving scars. Hilbert observed in a child of six months a varicellar ulcer on the lower lid and in the inferior part of the bulbar conjunctiva.

Treatment.—According to circumstances, incision of the vaccine vesicle, powdering with iodoform or moist boric, or sublimate gauze is indicated. As to treatment of secondary affections of the cornea, compare p. 208.

5. CROUP AND DIPHThERIA OF THE CONJUNCTIVA (PLATE V, FIG. 3)

These affections occur in laryngeal and faucial croup and in diphtheria, communicated either through the nose by dacryocystitis or through the mouth and nose by the patient himself.

Croup and diphtheria of the conjunctiva occur usually in isolated cases. Fatal infection of the nose, fauces, larynx, and trachea has often been observed as a sequel. The bacteriological examination in croupous conjunctivitis, which Saemisch sharply distinguishes clinically from diphtheria, has repeatedly shown virulent diphtheria bacilli. On the other hand, nothing but streptococci have been found in genuine diphtheritic inflammation. As a matter of fact, croupous conjunctivitis occurs comparatively often during diphtheria epidemics, and also before or after cervical and nasal diphtheria, with or without grave general infection of the patient or the persons around him.

It follows that true diphtheritic and diphtheroid attenuated processes may occur in the conjunctiva.

Croupous conjunctivitis often occurs in the wake of rubella, measles, scarlet fever, and smallpox. Again, diphtheritic changes of the lids and conjunctiva will occur, which may be distinguished from the typical clinical picture merely by the fact that the tissue infiltration is confined to circumscribed foci. Probable direct starting points of diphtherial conjunctivitis are eczema of the head, face, nose and lips, and lids; a most important means of communicating the secretion to the conjunctival sac is by the fingers of the children themselves.

Croupous conjunctivitis is almost always an infantile affection, beginning with acute eczematous catarrh, and sometimes accompanied by fever and swelling of the pre-auricular and submaxillary glands. The palpebral skin usually is soft and is more or less hyperæmic and œdematous. The temperature is usually elevated. On opening the lids, the transition folds protrude like ridges, and their surface, like that of the palpebral conjunctiva, is always covered by a smooth, bluish-white, light gray, yellowish-gray, or amber-yellow membrane. These membranes often encroach upon the intermarginal part of the lid, and can be more or less easily detached. The more recent the process, the more intimately does the deposit adhere to the conjunctiva. The tarsal conjunctiva underneath is dark red, rough, hyperæmic, and bleeds easily. At the onset, there is either no purulent secretion, or it is mingled with flakes and membranous shreds; after about a week, it is more plentiful, beginning with an arrest of the membrane formation. The bulbar conjunctiva is usually hyperæmic, often permeated with punctiform or splash-like hemorrhagias, chemotic, and may likewise be partly covered with membranes. The cornea is either clear or opaque, and shows a superficially diffuse, bluish hue. Other superficial, purulent infiltrates or ulcers of the cornea usually appear toward the end of the first stage, especially in cases where the bulbar conjunctiva also is coated with a croupous layer. There is often eczema of the lids, both before and after the affection. As a rule, the conjunctiva becomes normal after a week or two, but the membrane may continue for several weeks or even months. In these cases indurations have been observed over the entire conjunctival sac, completely covering the cornea, which may become destroyed.

If the coagulation spreads from the surface to the stroma of the conjunctiva, there will be pronounced diphtheritic conjunctivitis, with local elevation of temperature and considerable hyperæmia and œdema of the lids, especially the upper ones. These are sometimes as hard as a board, painful on touch, and can be everted with difficulty, if at all. Anæsthesia may be necessary to effect a thorough examination. Even

Healing takes place with varying degrees of cicatrization, producing posterior symblepharon, shortening of the lid fissure, entropion, trichiasis and secondary corneal affections, similar to granular conjunctivitis. The affection may also terminate fatally, especially in debilitated children, as, indeed, the general condition of the patient influences markedly the course of ocular disease.

In making the *differential diagnosis* the following condition must be eliminated: croupous conjunctivitis, occurring in the first stage of ophthalmia neonatorum, exanthematous conjunctivitis, pneumococcus conjunctivitis, with Koch-Weeks's infection, granular conjunctivitis, severe eczematous or oedematous catarrh, pemphigus, variola, herpes of the conjunctiva of the iris, traumatic conjunctivitis due to burns, and when foreign bodies lodge in the conjunctival sac for a long time. All these changes may, at the first blush, give the impression of croupous diphtheritic infiltrations.

In differentiating conjunctival croup from genuine diphtheria we should remember that the former, too, is highly contagious and not quite harmless—certainly not at the time of a diphtheria epidemic. Croupous conjunctivitis may change to conjunctival diphtheria. Nor is the result of the bacteriological examination always uniform, because even in conjunctival croup Löffler's diphtheria bacillus may be discovered, and in the clinically gravest forms of conjunctival diphtheria no bacteria but streptococci may be found.

As to prophylaxis, it should be remembered that chronic diphtheria has not always been recognized or correctly interpreted. It has been described as chronic croup, protracted diphtheria or recurrent diphtheria, and has been observed in tuberculosis, syphilis, scrofulosis, rhachitis, scarlet fever, and measles. The usual site of development is the faucial roof, at the posterior faucial section, and in the deep lacunæ of the faucial and palatal tonsils; a result, as a rule, of chronic catarrh of the upper respiratory tract and its sequelæ. This is notably true in hyperplasia of the palatal tonsils, of the lymphatic faucial ring and the faucial tonsils, tonsillar concretions, tonsillar and peritonsillar abscesses. These forms of diphtheritic infection are very favorably influenced by an incision of the tonsillar and peritonsillar abscesses, by enucleation of the tonsillar concretions and incision of the tonsils.

Where diphtherial conjunctivitis develops at an early stage of measles exanthem, perforation of the cornea and death in spite of the most careful treatment have been repeatedly observed, without any diphtheritic infection of the cornea or diphtheria bacilli in the fauces.

Treatment.—Croupous conjunctivitis should from the very first be treated as genuine conjunctival diphtheria, especially when there is an epidemic of diphtheria and when virulent Löffler's bacilli, streptococci, staphylococci, or pneumococci are found. One examination is not suf-

ficient. Patients should, therefore, be sent to a hospital and strictly isolated in a well-ventilated room. The nurse should be cautioned to observe the most scrupulous cleanliness, careful disinfection of the hands after touching the affected eye, destruction of material used for cleansing, frequent change of material used for painting, instillations, and bandages. Rubbing of the eyes in children is prevented by the measures described on p. 131.

It is important for the welfare of patient and nurse alike to have the oral and pharyngeal mucosa inspected frequently and to protect them by a gargling of peroxide of hydrogen, lime water, or diluted lemon juice. The latter is also administered internally as often as possible.

The irritative conditions in the first stage of the purely croupous inflammation are sometimes very violent, and are treated with dry boric compresses cooled upon ice. If the irritation is less severe, lukewarm boric compresses are well borne. The eyes are irrigated several times daily with pyocyanase, with tepid 3 per cent. boric acid or 0.9 per cent. solution. Should the motility of the lids be impaired and eversion difficult or impossible, the conjunctival sac is dried frequently with sterilized, spindle-shaped cotton pledgets, care being taken not to touch the cornea. The membranes are not drawn off, unless it can be done without injury to the conjunctiva. If, in spite of compresses and removal of the membranes, the disease remains unchanged, improvement may sometimes be effected by applying plenty of cooling ointment to the lids, and bandaging both eyes.

The catarrhal, blennorrhœal conditions of the second stage are often relieved by the careful removal of the secretion. Otherwise instillations of silver nitrate, 1 to 2 per cent., are made, or, if the secretion is very purulent, pyocyanase and zinc sulphate, $\frac{1}{4}$ to $\frac{1}{2}$ per cent., are applied.

As to circumscribed diphtheria, Wolfring advises rubbing yellow oxide of mercury into the infiltrated place during the first stage.

I have for years used frequent instillations of freshly-prepared lemon juice, diluted with purest glycerine, and a 1/10 per cent. solution of morphine in equal parts. This mixture easily penetrates into the diphtherial membranes, destroys the bacteria, and has merely an astringent effect. Painting with this mixture is still better. If the eversion of the lid is difficult, I induce alcohol-chloroform-ether anæsthesia (1 : 2 : 3) and paint the upper and then, if indicated, the lower parts of the tarsal conjunctiva and fornix. Contact with the cornea is to be carefully avoided, during treatment as well as during examination.

In confluent diphtheria, painting with lemon juice or instillation of it several times daily is again indicated. Vossius and Fuchs recommend painting with a 2 or 3 and even 4 per cent. solution of salicylic acid and

glycerine. Early instillations of 5 per cent. sodium benzoic and chlorine water have also been highly recommended.

Behring's serum has been used in appropriate doses in epidemics of croupous diphtheritic conjunctivitis without any material involvement of the cornea, and the transition of the disease from the first to the second stage was accelerated. It is also indicated by the general symptoms, and to prevent an extension of the infection to the nose and fauces in cases where the bacteriological examination reveals diphtheria or diphtheroid bacilli. However, the practitioner should not depend upon this alone, especially if he has not treated the case from the start, or if the cornea is even slightly involved.

The excretion of the toxin from the body may be quickened by inciting the skin function (*dialysé sudorifique* Golaz, etc.), as is done in cervical diphtheria. Copez recommends as local treatment the injection of a few drops of the serum under the conjunctiva. At the onset of the process, efforts should be made to prevent the increase of exudation by the regular application of ice compresses. As a rule, however, these measures come too late. This can be recognized by the fact that they are not borne well, and that the pain increases instead of abating. Cold then favors the advance of necrosis in the already anæmic upper strata of the conjunctiva and necrosis of the cornea.

In all cases, therefore, in which conjunctival diphtheria is no longer fresh and no longer in the ascendancy, the first stage should be transformed into the blennorrhœiform as speedily as possible. The secretion should be removed frequently and carefully, the conjunctiva irrigated with a 1 to 2 per cent. solution of hydrogen peroxide, and tepid compresses or fomentations, followed by warmer ones, should be applied. In diphtheritic infiltration of the lids the affected parts of the skin are frequently painted with a mixture of equal parts of filtered lemon juice, 5 per cent. benzoic potash, purest glycerine, together, if indicated, with instillations of atropine or eserine. Severe congestion is avoided by applying alternately compresses of warm and cold water, chamomile compresses, linseed cataplasms, and Langlebert's cataplasms.

Hot chamomile infusion also has a certain antiseptic effect, owing to the terpenes it contains. Linen compresses are immersed in the infusion, covered with fine flannel after application, and changed every few minutes like linseed cataplasms.

No more painting is done after the infiltrated parts have desquamated, but the application of heat is still continued, the temperature and duration of the application being gradually reduced. Instillations of cyanide of mercury 1.0 : 1500.0 or chlorine water, diluted with equal parts of distilled water, may also be made twice or three times daily until the scars have formed.

The treatment of the subsequent affections of the cornea is particularly important. If the corneal ulcers are rather centrally located, atropine is instilled; if peripherally, eserine. In impending perforation, artificial evacuation is indicated.

Adhesions of the lids to the globe should be prevented, so far as may be, by instilling potassium carbonate 1.0 : 30, and by repeatedly separating the agglutinations with a smooth, rounded glass rod, drawing the lids upward and downward.

Painting with silver nitrate should be avoided, if possible, in croupous conjunctivitis, as well as in diphtheritic conjunctivitis, even in the blennorrhœiform stage, as the granulations will usually cicatrize spontaneously. If, however, a few isolated spots should be very turgescent, or if the secretion should continue on an abundant scale, they may be carefully painted with a 2 per cent. solution of silver nitrate. If but one eye is affected, the other is protected by a watch-glass bandage renewed daily.

Eczema of the scalp, face, and lids must be immediately removed by careful cauterization of the eczematous places with a finely-pointed fused stick of precipitated white mercury ointment or zinc paste after cleansing from crusts and scabs. The same procedure should be followed with rhagades of the nostrils.

The sequelæ, consisting of extensive corneal opacity, shortening of the lids, etc., will have to be treated later by a specialist.

Postdiphtheritic paralysis, due to faucial diphtheria, etc., is extremely rare in diphtherial conjunctivitis. On the other hand, the corneal affections may not develop until long after the conjunctival process has subsided. An equally striking fact is the great irritability of the eyes, and the tendency to extensive cicatricial contractions, catarrhal and phlyctenular conjunctivitis.

6. PEMPHIGUS

So-called acute pemphigus, or febris bullosa, affects not only the new-born, but also older infants. Pergens observed in a year-old child a fatal course of the affection, with panophthalmitis of one eye and a large vesicle on the cornea of the other.

The *diagnosis* of chronic shrinking of the conjunctiva with vesiculation is difficult, as the clinical picture varies greatly and, owing to desquamation of the epithelial lining, vesiculation is less often observed. Accompanying the more or less violent conjunctival catarrh are ulcerous spots in the conjunctiva, which are covered with yellowish-white, whitish-gray or dirty green, croup-like and easily detachable deposits. According to Michel, the region of the inner canthus and the middle of the lower half of the scleral conjunctiva are favorite sites. Besides, fresh

vesicles and ulcers will develop in new places. The eruption is chronic, but there is neither disturbance of the general condition nor elevation of temperature. In the course of some months or years it leads to cicatricial contraction, which begins with the transitional folds. Xerosis of the tarsal and scleral conjunctiva follows, with ankyloblepharon, entropion and ectropion, trichiasis, cyst formation in the conjunctiva, and occlusion of the excretory ducts of the lachrymal glands. Cicatricial pannus degeneration, opacity, xerosis or ulcerous degeneration of the cornea, with partial or total loss of vision, are not rare complications.

The shrinking stage resembles the cicatricial degeneration of the conjunctiva that is due to chronic trachoma. As a rule, however, the shrinking is of a far higher degree. Corneal pannus is absent.

Pemphigus occurring only in the conjunctiva is very rare, and it is necessary to look for artefacts, notably erosions, to account for it.

Thus a young woman, to avoid work, had repeatedly put into her eyes a scrubbing soap which chemical analysis proved to be free alkali. This resulted in pemphigus-like manifestations which affected both eyes alternately, and led finally to entropion and symblepharon with subsequent diplopia. This was followed by dilatation of the lid fissure, operation for entropion, tarsorrhaphia medialis, and squint operation.

Prognosis.—The affection nearly always involves both eyes, even if not simultaneously; it also occurs with other skin diseases, as, for instance, prurigo, under the designation of “pemphigus symptomaticus.” The prognosis is unfavorable.

Treatment.—Internally: Arsenic; strychnine injected subcutaneously. Locally: To relieve the dryness of the eye, instillations of 3 per cent. boric solution, tepid glycerine, milk, olive or almond oil, or application of borated vaseline. The palliative methods used to restore shrunken parts and adhesions of the conjunctiva, as well as those usually employed to improve position anomalies of the lids or cilia, must be referred to the ophthalmic surgeon.

7. GONORRHOEAL OPHTHALMIA (PLATE V, FIG. 4)

The occurrence of blennorrhœa in the new-born is favored by conditions which occasion a prolonged or intense contact of the infantile eye with the pathogenic germs present in the annular constrictions of the genital canal, the lower parts of the cervi-canal, and the external orifice of the vagina (urethra, excretory duct of Bartholin's glands, fossa navicularis). This refers especially to retarded births, twin births, facial and brow presentation.

Early rupture of the amnion favors infection *in utero*, because the eyelids are easily everted, owing to the slow passage of the fetal head, which, being deprived of the protective fetal envelopes, allows patho-

genic factors to penetrate easily into the conjunctival sac. Or, the infectious secretion may be communicated to the fetal membranes by internal examination or, in facial and brow presentation, direct to the fetal face. In this way blennorrhœa with corneal infiltrates has developed antepartum, leading to bilateral destruction of the cornea. The forceps also may pull at the soft parts of the face in such a way as to provide a port of entrance to the pathogenic factor by opening the lid fissure.

The duration of the passage through the vagina is also to be considered. Thus, small children and those born by multiparæ run less risk than large ones and those born by primiparæ. Infants of the male sex run a greater risk, owing to the larger dimensions of the cranium.

A very mild form of blennorrhœal conjunctivitis which occurred twenty-four hours postpartum may be mentioned here as a curiosity, the infant being born within the fetal envelopes. Immediately after liberation, the eyes and face were carefully cleansed with sublimated cotton and freshly-boiled water. Four older children had been infected by blennorrhœa neonatorum (Plate IV, Fig. 4), each subsequent one in a less degree, and on the present occasion no gonococci were found.

The infection may occur immediately after birth; the secretion attaching to the cilia either spreads to the conjunctival sac on opening the lids, or it is communicated in the act of cleansing.

The great vitality of the gonorrhœal poison is etiologically important, as two, four or more years may intervene between the infection of the father and the birth of the gonorrhœal infant.

The period of incubation is two or three days at the most, the same as in gonorrhœal urethritis. Generally speaking, therefore, ophthalmoblennorrhœa occurring after the third day can be traced to a later infection. This has been observed as late as three or four weeks after birth and is occasioned either by the infected mother herself, through the lochia or mastitis (the latter especially when the infant sleeps in the bed with the mother), or more often through the instrumentality of the nurse, who attends first the mother and then to the infant. The usual carriers of infection are underclothing, sponges, and towels.

Infections carried from one infant to another, which were formerly observed in foundling institutions, lying-in hospitals, etc., can probably be accounted for in the same way. This is no doubt the reason why at periods of crowded deliveries in polyclinics the number of infections were particularly great. Communication from one eye to the other may occur during sleep, by secretion flowing from the infected into the healthy eye over the low bridge of the infantile nose.

It is a noteworthy fact that gonoblennorrhœa of the eye may cause gonorrhœal arthritis or gonorrhœal sepsis, or, by local extension, orbital phlegmons also. From a medical point of view it is important to note

that suppurative conjunctivitis of the new-born often presents the same clinical picture as gonoblennorrhœa, even if repeated bacteriological examinations of the secretion show no Gram-negative coffee-bean-like diplococci (gonococci), but other inflammatory and pyogenic factors, such as diphtheroid rods, streptococcus pyogenes, and bacterium coli. However, the course in these cases is usually more benign. The number of microorganisms varies according to the period of infection and the method of treatment. They may outlast suppuration, or increase as soon as treatment is arrested. Further experience will have to show whether, and in how far, there is an underlying trachoma in the new-born that are proved to be free from gonorrhœal infection.

Acute ophthalmogonorrhœa in older children, which is rather rare, is occasioned by gonorrhœal urethritis or ophthalmoblennorrhœa. If it is communicated by the child's own hands, the usual rule is for the right eye to be infected alone, or first. In little girls, vaginal blennorrhœa has been traced to attempted rape; in other cases, the infection was traced to the mother or to women who had suffered from vaginal blennorrhœa. Infection by bath-tubs and sponges has often been established.

The manifestations resemble those of gonoblennorrhœa in adults, there being swelling of the lachrymal glands, tendency to increased participation of the scleral conjunctiva, in the form of chemosis, increased stiffness and consequent danger to the cornea. At the onset of the disease the secretion inclines to coagulation and the formation of membranes.

A short time ago I observed a "house epidemic" in an infants' institute, where many nurslings and infants were cared for during the day, but in spite of diligent investigation the source of the infection was not discovered. Careful examination of all the inmates disclosed nothing but harmless catarrh of the conjunctiva with negative bacterial findings. Three little girls, aged two, two and a half, and six years, respectively, suffered from vulvovaginitis. Of six infected children, one of whom was a year-old boy, five were completely cured. The one first affected was a girl of twelve years, who, at the time of admission, showed considerable chemosis of the conjunctiva and swelling of the lachrymal glands. She was discharged with an adherent leucoma.

An affection of the conjunctiva which has been traced to benign vaginal catarrh, or other affections of the external genitalia, occurs as an endemic family disease of the conjunctiva, and has often been observed in school children. Its onset resembles gonoblennorrhœa in that there is considerable œdema of the lids and swelling and redness of the conjunctiva, but the secretion is scanty and usually in the shape of pus flakes which accumulate in the inner canthus or at the palpebral borders.

Its course, too, is less stormy, and the swelling of the pre-auricular lymph-gland is often absent. Occasionally, only one eye is affected, and the cornea is rarely endangered.

Course.—Aside from cases in which ophthalmoblennorrhœa is demonstrable at birth, the manifestations of catarrhal conjunctivitis set in between the first and fifth day, or later, according to the period of infection in one eye or both. The upper lid is somewhat swollen and hyperæmic, with elevated temperature. The eyes can no longer be opened normally, and seem to suggest coryza or weeping. The swelling of the lids increases during the next few days to such an extent that the upper lid can no longer be raised (Plate V, Fig. 4). On opening the lids, a thin, non-purulent secretion exudes, interspersed with grayish flakes and of a dark yellow color, due to blood-pigment. It sometimes coagulates into a glassy, amber-yellow membrane, covering in a uniform layer the light red, velvety, and easily bleeding palpebral conjunctiva, when the lids are closed. After another twenty-four hours the lids and their vicinity are so swollen that the rigidly infiltrated, hard upper lid droops like a ridge over the lower, less swollen lid, covering the lid fissure entirely. The palpebral skin is bluish-red throughout. The lid fissure can be opened only with difficulty and pain. Yellowish-red pus flows or spurts from the agglutinated lids. When the lids are everted, the transition folds protrude as broad, horizontal, reddish or reddish-gray ridges, the surface of which is at first very tense, smooth, and lustrous. The bulbar conjunctiva is strongly injected, permeated by striated and punctiform hemorrhages, with an œdematous yellow swelling, and is sometimes so distinctly raised from the sclera that it overtops the corneal margin like a circular wall.

The cornea, which had so far been smooth and lustrous, is now diffusely opaque, looking dull and gray like a tarnished pane. In cases running a rapid course, grayish-yellow, infiltrated places, with violent injection, appear before the climax is reached. In unfavorable or badly-treated cases they may spread rapidly to the deeper parts and into the area of the cornea, even though they look small and insignificant. The consequences are perforation of the cornea, prolapse of the iris, corneal scars, with or without adhesion to the iris, and anterior, central, capsular cataract. There may also be staphylomatous protrusion of the cornea and retina, perhaps in the gravest cases with loss of the lens, and panophthalmitis—by the infection spreading to the uvea—and eventual shrinking of the eye.

If the affection takes a less acute course, the corneal infiltration may become demarcated and the scars may heal, leaving such minute traces that later they can be seen only with the loupe and ophthalmoscope.

Desquamation of the epithelium, which initiates suppurative infil-

tration of the cornea, begins either in the centre or lower central portion of the so-called lid-fissure zone, or at the periphery.

Infection is favored by the defective nutrition of the cornea generally, perhaps also by the pressure exerted by the non-resilient palpebral borders upon the corneal centre. The corneal periphery is endangered by the retention of secretion in the recesses between the corneal margin and the overhanging wall of the bulbar conjunctiva.

The general condition is usually undisturbed, aside from restlessness and a transient rise of temperature. In prematurely-born or artificially-fed children there is also considerable loss of weight.

The affection persists in this stage from three to six days, according to the degree. The secretion becomes more viscid, yellowish or greenish-yellow, and so abundant that when the lids are opened the pus wells out. This is followed by a reappearance of the normal folds on the lids. Swelling of the lid and chemosis of the bulbar conjunctiva subside, and the little patient may open his eyes for at least a short time in the evening. The folds impart an uneven, granulated or irregularly striated appearance to the mucosa of the lids and transition folds. The decrease of the pus does not keep step with the decreased swelling of the lid and conjunctival hyperæmia. On the other hand, the inflammatory manifestations gradually recede, so that the lid borders and cilia are only occasionally glued together. This is more marked in the morning and the purulent secretion is more noticeable at the inner canthus. After about three weeks the lid fissure may again be opened more freely.

The duration of the disease is from three to five weeks, but may persist from six to eight weeks, especially in illy-nourished and underdeveloped infants.

In serious cases, which have been treated incorrectly or not at all, large papillary caruncles of the conjunctiva may develop. These may gradually be made to disappear, but if they are complicated by some form of diphtherial infection, or if cautery has been wrongly applied, conjunctival scars will persist.

Complications by phlegmonous abscesses of the lids and the subconjunctival connective tissue are very rare, but they may also be caused by gonococci. The acute swelling of a joint, or acute polyarthritis, are both rare, either as concomitant manifestations or as sequelæ.

The *prognosis* is generally favorable, if the cornea is still unimpaired at the beginning of the treatment; the later the affection sets in, the more favorable will be its course. It is also favorable with correct hospital treatment and with a good general state of health.

Nevertheless, a cure should never be promised even in apparently favorable cases of blennorrhœa neonatorum, although the probability of a cure may be held out.

Not infrequently children are taken to the physician after the affection has existed for weeks or months, and one or both eyes have already been injured or destroyed.

The prognosis is unfavorable in premature births. Especially is this true in weak infants who cannot take the nipple well, who are fed on artificial food, have an icteric skin, or are otherwise unhealthy. Fortunately, these infants usually die during their first few weeks. There is often slight swelling of the lids and suppuration of the conjunctiva, complicated by keratomalacia with ulceration of the cornea, and subsequent shrinking of the eye. Here medical aid is nearly always powerless.

Differential Diagnosis.—In panophthalmitis, which may likewise be associated with inflammatory oedema of the lids and chemosis of the conjunctiva, the globe protrudes and its motility is impaired. Blennorrhœa or copious, purulent secretion is absent.

In blennorrhœa of the lachrymal sac and the nasolachrymal canal, due to occlusion of the lachrymal duct (see p. 106), the concomitant manifestations of conjunctival blennorrhœa (oedema of the lids, etc.) are absent. The mucopurulent secretion is usually confined to the inner canthus, or the lower parts of the conjunctival sac.

The cornea remains uninjured, in spite of the fact that the affection may have persisted for months. Hyperæmia and chemosis of the conjunctiva will disappear after the occlusion has been removed. Besides, this form of blennorrhœa is mostly unilateral, and affected infants are often not presented for treatment until many weeks or months after birth.

Prophylaxis.—Infants delivered by midwives still run a risk of purulent inflammation of the eyes. It is necessary to instruct these women very carefully how to cleanse the eyes and carry out Credé's prophylactic method. They must be taught to proceed with the greatest care, with thoroughly disinfected hands, and to instil the fluid with an absolutely clean glass rod. They must be instructed to call a physician immediately if, in spite of precautions, the conjunctiva should manifest inflammatory signs.

A law has recently been enacted in Bavaria providing for the compulsory registration of blennorrhœa neonatorum, and giving the medical officer the right to institute protective measures. He is also entitled to investigate the method of infection, and whether the midwife has done her duty.

Furthermore, if a midwife recognizes the presence of leucorrhœa during the period of pregnancy, she should insist upon an immediate medical examination, so as to make sure whether the affection is gonorrhœal. Many pregnant women do not know they are suffering from an infectious vaginal disease.

The lay public should also be instructed. This was first carried out in Le Havre, where persons registering the birth of a child are given instructions and warnings in plain language. Roth, Saemisch, and Widmark have compiled similar instructions. Widmark's directions, in Sweden, have been embodied in a calendar which is published by the Stockholm Academy in an edition of 500,000 copies, and contains each year information on some important subject. It is due to the publication of these instructions, which appeared in 1885, that in 1890 only twenty-nine cases of ophthalmia neonatorum were treated in the hospitals, as against ninety-nine in 1884. Stieler and H. Cohn suggest that the number of blind individuals annually admitted to institutions be published, and the proportion of that number who have become blind from gonorrhœa. Cohn's demand that men suffering from gonorrhœa, be the remaining traces ever so slight, should abstain from sexual intercourse, is probably rarely heeded, and it is, therefore, important that vaginal and urethral catarrh, which may be due to the gonococcus, should be removed during pregnancy. Furthermore, in every suspicious case the genital tracts and the external genitals of the parturient woman should be cleansed at the beginning of delivery, in the manner recommended by Kuestner. This is as follows:

If there is still time, the prospective mother is given a warm bath. While in bed, her external genitals are lathered with soap. This is removed with warm water, and the parts are irrigated with a sublimate solution, 1 : 4000, and rubbed with a cotton pad immersed in a sublimate solution of 0.1 : 1000. Soap is now applied to the vagina with a new toothbrush, which can enter all the crevices better than a cotton pad. The foam is removed by turning the brush and irrigating with a sublimate solution of 1 : 4000. This procedure should be observed after every internal examination. No more examinations should be made than necessary, and only after the examining hands and arms have been thoroughly disinfected. Irrigation is then repeated with a 0.5 : 1000 sublimate solution.

Kuestner also recommends cleansing the eyes of the new-born immediately after the head has been delivered and before the shoulders have presented, all the mucus attaching to the lids being carefully removed with small cotton pads, which are kept in readiness in an iodine trichloride solution of 1 : 4000. Care should be taken not to have the eyes opened during the procedure.

Credé's method is as follows: Immediately after delivery, and, if possible, before the umbilical cord has been detached, the lids are slightly drawn apart with two fingers. A round glass rod, 5 cm. long and 3 mm. thick, and smooth at both ends—not a dropper—is used to let a single drop of a 2 per cent. silver nitrate solution fall upon the centre of the

cornea, the latter being barely touched by the rod. Instructions as to the care of the rods have been given on p. 132.

No further inspection of the eyes is made. Nor is the instillation to be repeated for twenty-four or thirty-six hours, even if there should be a slight redness or swelling of the lids and mucous secretion.

In infants born of notoriously hæmophilic parents there is danger of fatal hemorrhage from the free surface of the conjunctiva. This has happened after a prophylactic instillation of $\frac{1}{2}$ per cent. silver nitrate solution. In these cases, therefore, calcium permanganate 0.1 : 1000, which is chemically more indifferent, should be applied.

During the first bath, the infant's eyes are not allowed to come in contact with the bath water.

Silver nitrate destroys the gonococci present in the secretion and on the surface of the mucosa, the caustic scab at the same time forming a protective layer against a fresh invasion of gonococci.

Until recently, optional instructions reading as follows had been in force in Bavaria: "In all cases of purulent vaginal discharge, one drop of a 2 per cent. silver nitrate solution must be instilled into each eye immediately after the new-born infant has been cleansed." Midwives, however, rarely, if ever, paid attention to them, knowing, as they did, that silver nitrate, if not fresh or if concentrated by evaporation, irritates or injures the eye. Injurious effects have also been observed by the instillation of larger quantities than the one drop prescribed by Credé. Grave suppuration has also occurred in the eyes of prematurely-born infants, or those with arrested development, probably due to diminished resistance of the epithelial covering of cornea and conjunctiva.

Since compulsory prophylactic instillation in institutions has reduced ophthalmia neonatorum to 0.1 per cent., this treatment has now been made obligatory in Bavaria. The state regulations require midwives "to instil into the eyes of all new-born, before the umbilical cord has been detached, one drop each of a 1.2 per cent. solution of silver acetate, which they must carry with them in a blue eye dropper."

Silver acetate, which has been recommended by Kroenig and Zweifel, has the advantage of remaining in constant solution, since no more than 1 per cent. is dissolved at ordinary room temperature, and any precipitated crystals become covered with silver chloride, thereby losing their caustic property. Moreover, the irritation of the conjunctiva from acetic acid is slight, as compared with that of nitric acid.

Other substitutes, such as albargin, argentamin, argonin, argyrol, ichthargol, itrol, kollargol, largin, protargol, sophol, syrgol, etc., have not been included in the compulsory instructions, because their bactericidal and germ-arresting effect is considerably less than that of silver nitrate, even though they may be less irritating and penetrate deeper

into the tissues. Moreover, some of them leave much to be desired in subtlety of preparation and stability. They may also lead in time to argyrosis of the conjunctiva and cornea.

Sophol, 5 per cent., has proved its efficiency in the Women's Clinic of Breslau, where, according to v. Herff and Gallatia, there was only one late infection among 1519 infants, and this was almost completely cured in four days. Early infections were reduced from 0.26 to 0.12 per cent. (Gratowski). F. v. Arlt recommends cusylol (cuprum citricum) as a prophylactic preparation free from all danger.

If there is no cause for suspicion on the part of mother, midwife, and nurse, which, of course, happens very often, the Credé prophylaxis may be dispensed with.

Late infection can be prevented only by scrupulous cleanliness and protection of the infantile eye from infectious contact, especially during washing and suckling. The fingers of an infected mother, soiled with vaginal secretion, and the hands of a nurse who has been attending other lying-in women or infected infants, are particularly dangerous. An infant infected with blennorrhœa must, therefore, be isolated and entrusted to a nurse who will not come in contact with healthy children at all, and with the mother only when putting the infant to the breast. Rigid separation of dressings is important. A case is known where a nurse cleansed the infant's eyes with a piece of cotton which had lain in front of the infected mother's vagina. Wet-nurses suffering from blennorrhœa of the eyes or vagina must not suckle infants.

Mild cases of conjunctival catarrh, occurring postpartum, may invite late infection, unless they have been correctly diagnosed and competently treated. They are usually cured in the course of a few days by cold compresses or instillation of light astringents. If the suppuration is extensive, cauterization will be necessary. If complicated by swelling of the lids and a bacteriological demonstration of gonococci, the same treatment should be instituted as in ophthalmogonorrhœa. Should the latter set in, it is necessary for the physician to inspect the eyes at least once a day. He should also convince himself that the nurse carries out his directions correctly.

Separate washing utensils are required. To protect the bed clothes, a clean linen cloth is placed under the infant's head, frequently changed, and immediately after use folded well together for the laundry. The infant's hands are loosely fastened by a towel which is pinned toward the back, so that he can not touch his eyes, although the arms are freely movable. A protective or watch-glass cover for guarding an apparently healthy eye will not keep in position over an infant's eye, and is therefore useless, but the bridge of the nose should be anointed with a little borated vaseline. The healthy or less infected eye is always cleansed first with steril-

ized cotton, and it should also be protected by a daily instillation of 1.2 per cent. acetate of silver, in the manner prescribed by Credé.

The secretion is carefully wiped off with small cotton pads which, after use, are placed on paper in glass dishes. This is folded up without touching the cotton pads again, and immediately burned. The lid fissure is now slightly opened, taking care to avoid all pressure on the eye, and the conjunctival sac is carefully and gently cleansed with tepid physiological salt solution or borizin (equal parts of borax 3 per cent., boric acid 3 per cent.) which is kept in a small glass container (Undine). The conjunctival sac is irrigated with the same solution by alternately opening and closing the lid fissure, with the point of the glass container as closely as possible to the eye, carefully avoiding injury to the corneal epithelium.

This procedure is repeated every one or two hours, day and night if necessary. If possible the infant's sleep should not be disturbed. The nurse attending to these matters should always wash her hands thoroughly with soap and warm water after each application, including her finger-nails, which should be trimmed short.

When there is copious secretion, there should be no bathing, as it would appear that catching cold (?) may induce an exacerbation of the condition.

The physician, as well as the nurse, should wear protective spectacles when treating the infected genitals and eyes, so as to guard against spurting pus. The spectacles are washed in a 1 : 1000 sublimate solution immediately after use. Should the slightest trace of the secretion enter the eye, it should be immediately cleansed with sublimate, 1 : 5000. A few drops of the solution should be instilled into the eye, or, better, a 2 per cent. silver nitrate solution, provided it can be procured without delay. After the instillation, cold water, or preferably ice compresses, are to be applied uninterruptedly for several hours.

This method of treatment was first systematically carried out by Lamhofer, and has proved its value in cases where the lids had become turgid and the cornea had a grayish, opaque appearance.

A daily instillation of a $\frac{1}{4}$ per cent. physostigmine solution often diminishes the secretion in a striking manner. The best time to make the instillations is in the forenoon.

The attention of relatives and nurses should be called to the great danger to the children and the great risk of contagion for themselves, unless all precautions are implicitly observed.

There is a case on record of an assistant at an ophthalmological clinic who died of sepsis, after his right index-finger had become infected by the pus of a blennorrhœal infant.

Kissing infected infants should be rigidly forbidden.

In losses of corneal substance, Burchardt recommends application of silver nitrate in the following way: The physician holds the infant's head as described on p. 63, bending it back with a slightly lateral inclination, so that the inner canthus is the lowest point in the region of the affected eye. An assistant applies a tepid 1/10 per cent. silver nitrate solution in drops or larger quantities to the inner canthus, while the physician's right thumb rapidly works the lower lid up and down at the rate of several times a second. At the same time, the left index or middle finger slowly moves the upper lid laterally to and fro. The speed of the movement imparted to the lower lid is so great that the fluid, as it is instilled, may spurt as high as two inches, and is effectively retained between the upper lid and globe. The massage necessarily given thereby quickly obliterates the tension of the globe, and even softens the upper lid sufficiently to allow the fluid to pass to the upper transition fold. When this manipulation has been continued for about half a minute, the conjunctival sac is so clean that the silver nitrate solution returns clear. The procedure is applied four times daily, taking care that no pressure is exerted upon the eye, especially when there are corneal ulcers. Between the applications, tepid compresses of a 5 per cent. mixture of chlorine water are applied. Any fibrinous membranous deposits on the palpebral conjunctiva must be previously removed with sublimated cotton tips, the lid being everted for the purpose.

Burchardt effects eversion of the lid as follows: The skin of the brows is kneaded in below the upper orbital margin, while the finger-tips, working alternately, hold the skin of the upper lid upward. A rapid movement imparted to the lower lid at the outer canthus pushes the lower lid under the upper one, which is thereby everted. As compared to the ordinary method of eversion by traction at the palpebral border of the cilia, this procedure has the advantage of keeping the lids from contact with the cornea.

This treatment, however, can be given only in clinics where there is a trained staff. If working single-handed, the physician gently paints the everted conjunctiva of the lids and transition folds once daily with a 2 per cent. solution of silver nitrate. This, however, should not be done until suppuration commences, as the slightest previous irritation of the eye is injurious. If this procedure is borne well, causing the suppuration and turgescence of the conjunctiva to diminish, the solution is applied in gradual dilutions down to 1½ and 1 per cent.

The conjunctiva of each lid is painted separately. This will cause the transition folds, which are most in need of treatment, to become more prominent. The cauterization may also be graduated by painting the conjunctiva of one lid at a time, or by allowing the solution to act upon the conjunctiva for a shorter or longer period before rinsing it

with water. This procedure is repeated every forenoon at the same hour until the purulent secretion disappears. When necessary the application may be made twice a day, but not until the scab has disappeared and the epithelial layer of the conjunctiva has undergone regeneration.

To relieve the pain caused by the cauterization, ice compresses of absorbent gauze are applied for fifteen to thirty minutes, without exerting any pressure. To remove the profuse secretion after cauterization, the lids are slightly opened, and the pus removed with sterilized cotton pads, without wiping. These pads are immediately wrapped up in paper after use and burned.

As the secretion and turgescence of the mucous membrane decrease, the strength of the solution is gradually reduced to 1, $\frac{1}{2}$ and $\frac{1}{4}$ per cent.

The $\frac{1}{2}$ per cent. solution should be freshly prepared every week, the 1 per cent. solution every two weeks, while the 2 per cent. solution will remain stable for a longer time. Two small glass dishes are required for the painting process, one containing the silver nitrate and the other the salt solution. They, as well as the brushes, must be properly cleaned after every application, and kept in a covered glass vessel. The brushes should be about 7 to 8 centimetres long, and a special set is required for each patient. Upon termination of the treatment they are burned. If the glass dishes are to be used over again, they must be sterilized immediately before use. If several patients are treated at the same time, the dishes for each are kept in separate glass vessels, labelled with the names of the respective patients. The vials containing the silver nitrate should also be labelled and kept separate.

In the presence of peripheral corneal ulcer, physostigmine ($\frac{1}{2}$ –1 per cent.) is instilled; while in central ulcers, atropine ($\frac{1}{2}$ per cent.) or scopolamine ($\frac{1}{5}$ per cent.); the first application being made an hour after painting; thereafter two or three times in the afternoon, at equal intervals.

Moist compresses of tepid chlorine water (1 : 2–1 : 3 aq. dest.) promote the healing of the ulcer; their temperature is gradually increased as the secretion is reduced.

When there is extensive destruction of the cornea, prolapse of the iris, or keratomalacia, a specialist should be immediately called in.

The general nutritive condition of the infant is of paramount importance in ocular affections, as is shown by the fact, amongst others, that illegitimate infants are oftener affected by this and other diseases than legitimate ones. Want of cleanliness, aphthæ of the buccal cavity, premature weaning, omission or careless use of the Soxhlet apparatus, may conduce to an exacerbation of an apparently cured ocular affection.

Intercurrent profuse diarrhoea is sometimes accompanied by abrupt diminution of blennorrhœa, but the reverse effect has also been observed.

H. Cohn established the postulate that a lying-in woman whose infant shows signs of inflammation of the eyes should not be discharged from the institution.

Non-specific blennorrhœa neonatorum cannot always be distinguished from the specific form, even by bacteriology, and the physician will do well, therefore, to treat every case as specific.

There should also be careful and competent treatment of any gonorrhœal infection of the genitals, anus, and articulations, as well as of any other disease that may be present.

Many other methods of treatment have been recommended.

Bernheimer found airol powder superior to silver nitrate "at least at the onset of suppuration, and as long as there is considerable turgescence or suppuration." After carefully cleansing and rinsing the affected eye, the powder, in quantities varying with the prevailing conditions, is applied to the mucosa of the lower lid, which has been everted as far as possible, with a glass spatula, two to four times daily. After the powder has been converted into pap, covering the everted eyelid, the latter is very gradually allowed to return to its normal position; the upper lid is simultaneously raised, so that the entire mass may penetrate into the conjunctival sac. Gauze pads, immersed in boiling water, are applied while warm and wet. A few granules of airol powder are scattered into the healthy eye for a few days, for prophylaxis. After the secretion has abated or ceased, a 1 per cent. solution of silver nitrate is instilled once or twice daily, to prevent relapses. This method of treatment has also effected cures in adult cases of advanced blennorrhœa within one to two weeks. Bernheimer attributes these favorable results to the fact that the powder remains in the conjunctival sac for several hours, "so that the liberated iodine acts uninterruptedly upon the gonococci."

A. Voigt recommends instillation twice daily of argentamine for treatment, and of atropine for prophylaxis. The palpebral border is to be cleansed with vaseline several times daily, scrupulously avoiding touching the cornea, and is to be irrigated to prevent eczema.

Adam applies once or twice daily, according to the gravity of the case, 1 drop of a $\frac{1}{2}$ -1 per cent. solution of silver nitrate to the conjunctiva of the everted lid, and instructs those in charge to wipe off the secretion every hour as it exudes from between the lids, followed by blenolenicet ointment (10 per cent.) introduced into the conjunctival sac. F. Schoeler agrees that this treatment arrests the secretion, and protects the cornea from injury. He uses the ointment (5 per cent.) during the night only, assuming that members of the family cannot be

expected to cleanse the eye thoroughly several times daily, if the lids are covered with ointment and have a smooth surface. He also paints the conjunctiva of the everted lid with a 2 per cent. solution of silver nitrate in the manner described; then, "during the day, cold compresses, with or without ice, irrigation with chlorine water, permanganate of potassium and similar solutions; instillations, once or twice a day, of 5 per cent. argyrol, which may be occasionally replaced by protargol or sophol."

F. v. Arlt is in favor of eusylol, using a 2 per cent. solution in mild cases, and a 5 per cent. solution in graver forms of blennorrhœa neonatorum, whether gonococci have been demonstrated or not.

The *treatment* of acute infantile gonoblennorrhœal conjunctivitis is in a general way the same as in gonoblennorrhœa neonatorum. It presents greater difficulties when there is marked chemosis of the bulbar conjunctiva, for the reason that weak silver nitrate solutions are useless, while stronger ones increase the chemosis and, consequently, conduce to necrosis of the cornea. Painting with silver nitrate (2 per cent.) is therefore not permissible until the tense and chemotic conjunctiva has become soft and succulent, and the swelling and membranous deposits or gray infiltrations have disappeared. Consequently, often nothing remains at first but to order rest in bed, and careful cleansing as described on p. 162. This should be done every half-hour, day and night, and later every one or two hours, according to indications.

If the turgescence of the lid persists, interfering with thorough cleansing, external canthotomy may be necessary. This will at the same time relieve the pressure exerted by the lids upon the cornea.

For this purpose, the outer canthus is stretched tight, drawing the temporal part upward and downward with the fingers as far as possible. Incision is then made with a pair of scissors, held horizontally, the blunt blade being advanced in a straight direction behind the canthus, until the resistance of the osseous orbital margin is encountered. The wound should be thoroughly exsanguinated, by separating the traumatic edges from time to time. Healing of the wound is left to itself. It is important in this operation to make the incision sufficiently long and deep.

Continuous irrigation (Paulsen) is also to be commended.

A pail of freshly-boiled water is placed on a chair, a rubber tube of 6 to 8 mm. diameter, and weighted with a metal plate, acting as a siphon. The temperature of the water need not be less than 12–15° C. (54–59° F.), and it may be gradually raised to suit the comfort of the patient. At first a pailful of water is allowed to run over the closed lids hourly, day and night; later, the interval is two hours. The effect is immediate. If the patient opens his eyes from time to time every trace of pus is washed away. The œdema of the lids and conjunctiva is visibly de-

creased; the drooping upper lid can be raised and the lid fissure opened with less difficulty. All this is probably due to the contraction of the palpebral and conjunctival vessels, which had become relaxed and inefficient. The reduction of the chemosis increases the blood supply to the corneal periphery; the purulent conjunctival secretion in the recesses between the corneal margin and the overhanging wall of the chemotic bulbar conjunctiva is removed and renewed accumulation prevented. Frequency of application depends upon the intensity of the process. In the presence of extensive ulcers or prolapse of the iris, the flow of water is reduced in quantity and force by lowering the pail, but the duration of the flow is correspondingly increased.

The nursing staff must be repeatedly shown the technic of this procedure, as it must frequently be adjusted. To prevent contact with the cornea the aperture of the tube should be at a suitable inclination.

Gepner and Wolffberg have obtained good results in a few cases by using irrigation of the conjunctival sac with formaldehyde. Gepner prescribes 1 : 1000, later 1 : 2000, every two hours. Wolffberg allowed a solution of 1 : 500 to flow for three to five minutes, twice daily.

Here, again, the use of physostigmine and atropine or scopolamine (see p. 165) is very valuable when there are corneal infiltrations.

Silver nitrate, used in the manner described on p. 163, should not be applied until the lids have lost their hard tension, until the mucosa forms ridges and grooves, and there is profuse mucopurulent secretion and the chemosis of the conjunctiva has completely disappeared.

Burchardt's method of eversion is the best for this purpose. If the lids are difficult to evert, they are painted separately, first the upper lid, letting the patient look down, then the lower one while the patient looks upward. The latter part of the procedure is the easier, as patients have a tendency to look upward during the painting process. If necessary, the lids must be held in the everted position by an attendant, all pressure on the eye being carefully avoided.

Grayish discoloration of the conjunctiva (argyrosis) may occur in the course of prolonged treatment, even with weak solutions of silver nitrate, but this is remedied by painting with zinc sulph. or copper albuminate (0.2 : 20). These applications should be replaced by others, if the reaction of the mucosa becomes insufficient.

Accidental presence of corneal pannus is an excellent protection against necrosis of the cornea, and, after the conjunctival gonorrhœa has subsided, the pannous parts of the cornea have often cleared up.

In two cases of grave conjunctival gonorrhœa, with simultaneous presence of gonorrhœa, Knies prescribed copaiba (twice to three times daily, four to six capsules), and observed that from the fourth day the conjunctival infection took a surprisingly mild course.

The posture of the patient should always be such as to prevent the secretion from flowing over into the healthy eye. Further protection is provided by prophylactic instillations, as described on p. 158, or the eye may be protected by a fairly large watch-glass, fastened to the surrounding skin with collodion and cotton, or adhesive plaster (Fig. 27). This is renewed daily, preceding the inspection of the affected eye, until the secretion has been arrested.

Separate, labelled glass dishes, vessels containing dressings and cleansing solutions (boric acid, sublimate, or weak wine-red potassium permanganate) should be provided.

FIG 27



Watch-glass bandage.

Metastatic, abortive gonorrhœal conjunctivitis resembles catarrhal conjunctivitis, but is confined principally to the transition folds. The secretion is usually slight, and papillary proliferation rare.

Treatment.—Ice compresses; internally, 0.1–0.3 Gm. of quinine sulphate a day.

As to complications with diphtheritic infections, see p. 146; for the treatment of phlegmons of the lids, p. 79.

It very rarely happens that so-called chronic blennorrhœa persists after the cure of gonoblennorrhœa. It is characterized by thickening, velvety consistency, and redness of the conjunctiva, notably of the transition folds. It is treated by applying at two-day intervals a well-smoothed stick of sulphuretted or aluminated copper to the palpebral conjunctiva. This, however, should not be done until cicatrization of corneal ulcers—if any

—has distinctly commenced, but in no case where they still display a purulent coating. The first application is attended by exquisite pain.

Papillary cockscomb-like proliferations, occurring in grave cases which have been treated incompetently or not at all, are dealt with in the same way. They can be recognized later by minute scars in the conjunctiva.

If gonorrhœal conjunctivitis has not been thoroughly cured, the affected eye may subsequently be destroyed by panophthalmitis, even after the lapse of years. Fresh suppurative inflammations may occur in a conjunctiva which has not resumed its normal condition, or the globe may be infected from a corneal scar. Absence of gonococci or pyogenic factors should, therefore, be established by microscopic examination or cultures before patients are definitely discharged. A solid outward demarcation of corneal scars with anterior synechiæ is, therefore, of particular importance. Patients and their relatives should be urged to seek medical aid immediately upon the slightest irritation of the eye.

Artificial eyes, which are sometimes worn after gonorrhœal infection involving shrinkage of the eye, may irritate the conjunctiva and cause a subsequent affection of the healthy eye. The most scrupulous cleanliness is, therefore, required in treating, inserting and removing the artificial eye, and a new prosthesis should be procured as soon as the old one is no longer well tolerated.

Clearing up of the corneal scars should not be attempted until the secretion has been completely arrested. Tincture of opium, which is usually instilled in these cases, is contra-indicated in the new-born.

The Supreme Court in Germany has decided that gonorrhœal infection of an attendant, contracted from a venereal patient, entitles to damages. Some gonorrhœal secretion had invaded the plaintiff's right eye, which became blind. "A purulent inflammation of the eye, caused by gonorrhœal infection, is not to be regarded as an infectious disease, if the infection is confined to a limited area and does not spread to other parts. This was a case of impaired working ability, caused without the coöperation of other factors, by an injury that occurred independently of the will of the injured, by a sudden external mechanical cause, and was made possible by the fact that complainant made an involuntary movement to the eye with a hand laden with gonococci."

8. TRACHOMA

Trachoma (conjunctivitis *granulosa*) occurs in infancy. It is not yet definitely established that it is due to a specific cause. Infants under three months are not attacked, possibly for the reason that in the first months of life there is no adenoid tissue. The fact that children under three years do not mingle with others as much as adults may explain why they are not more frequently affected. Infection is favored

by lowered economic conditions, overcrowding, and promiscuous use of all kinds of articles. Older children are predisposed to the disease when there are manifestations of obvious scrofula, tuberculosis, and impaired general resistance. On the other hand, hygienic measures seem to destroy the pathogenic factor outside the human conjunctiva.

Persistent unilateral trachoma is rare, and it is doubtful whether this form is not due to injury, dust or ashes, or contusion.

In strongly-infected districts the disease usually attacks all the members of a family, it being an exception if one of them escapes; although, according to Junius, in families living under better conditions, the infection is confined to a few members. Trachoma imported from an infected to an immune district may be as severe as at the original place of infection, though it may run a milder course.

It has been stated that trachoma is dependent upon certain telluric and climatic influences, and that it usually occurs in swampy, low-lying districts and in the valleys of sluggish rivers, rather than in districts of higher altitude and those with rapidly-flowing rivers. This is supported by the fact that in districts where there is a favorable culture ground for the breeding and preserving of miasms of all kinds trachoma is usually present in a grave, endemic and epidemic form, while at an altitude of 1700 feet and upward it is much rarer, of sporadic type, and runs a milder course. Work in the open air is not an unconditional protection, since dust and the rays of the sun irritate the conjunctiva, and invite infection by frequent wiping of the eyes. Thus in 1868-1869, among the recruits rejected on account of trachoma in the Bavarian province of Upper Franconia, the largest contingent (150) was supplied by the rural population. Next ranked those working in closed rooms (114). In the rural district of Hof, with its scattered villages lying at altitudes of 1628 to 2198 feet, eleven out of a hundred and forty-four recruits (8.09 per cent.) had to be temporarily rejected in 1868, and fifty-three out of a hundred and sixty-two (32.71 per cent.) in 1869.

So there is no assurance that non-trachomatous districts like the Bavarian plateau (over 1700 feet), which seem to have a favorable influence upon trachoma, will remain immune if populated for many years with large numbers of rural laborers immigrating from such countries as Polish Russia and Upper Italy, many of whom suffer from trachoma. The best protection is the rigorous exclusion of foreigners suspected of trachoma, as is done in the United States. Grave results may follow from deep invasion of a corneal affection, rendering a considerable number of patients temporarily incapable of work, and the disease may gradually or even suddenly spread over vast areas, inflicting considerable economic damage.

It is rarely possible to determine its onset. It nearly always runs

a chronic course, and in grave cases is often accompanied by swelling of the regional lymph-glands. In some cases it sets in with grave irritation of the conjunctiva. This may be due to a mixed infection of preëxisting trachoma with pneumococcus conjunctivitis or with the Koch-Weeks bacillus, or it may be "genuine acute trachoma." This acute form often undergoes a relatively rapid cure under competent treatment, as was observed, for instance, in physicians whose eyes became infected by spurting pus while expressing granules.

It is still undecided whether, in chronic trachoma, the granules develop first and the inflammatory reactions later, with diffuse infiltration and thickening of the surrounding mucosa, or whether the reverse is the case.

The transition folds are principally affected, the plica semilunaris only exceptionally.

The granules increase in number (Plate V, Fig. 6), particularly in the nasal and temporal parts of the transition folds, where the arcuate end of the tarsal border approaches the free palpebral border. Simultaneously, there is considerable thickening of the posterior margin of the tarsus which, together with increased diffuse infiltration of the internal tarsal surface, forms a pronounced folding of the conjunctiva, which assumes a dirty, livid appearance.

As the granules and infiltration extend into the deeper parts and along the surface toward the tarsobulbar conjunctiva, plica semilunaris, and tarsal cartilage, pseudoptosis develops. This is an important diagnostic point. It is caused by the inability of the levator palpebrarum to carry the increased weight of the upper lid, whose tarsus is three or four times thicker than that of the lower one; or it may possibly be due to infiltration of the smooth musculature adjacent to the levator tendon.

Scanty secretion of a mucopurulent consistency points to slow development, profuse secretion to a rapid progress of the pathological changes of the conjunctiva. The affection is spread by the latter class of cases.

Pannus of the cornea (Plate VII, Fig. 2) occurs in nearly one-third of the cases, usually after the lapse of a year, rarely sooner. It manifests itself along with the ciliary injection of the upper corneal margin, and often keeps step with the progress of the conjunctival affection. It usually disappears with the removal of the granules, especially those of the upper lid. It begins at the upper corneal margin, spreading toward the lower one. On the other hand, pannus due to tubercular infection may originate at any part of the corneal limbus. A broad strip of the bulbar conjunctiva adjacent to the limbus is nearly always uninvolved in the pannus of trachoma.

Epithelial erosions and granular elevations, which extend into the corneal tissue beyond the lower margin of the pannus, indicate the invasion of the cornea by the disease. In like manner the superficial infiltration of the membrana elastica anterior and the anterior corneal layers (often rendered visible by fluorescein staining) predicate corneal involvement. If the granular infiltrates descend to the lower half of the cornea, new-formed vessels shoot forth from the lateral and lower parts of the limbus.

The pannus is either thick and highly vascular, or thin and slightly vascular (pannus crassus or carnosus; pannus tenuis or siccus). Both are associated with a diffuse cellular infiltration of the affected corneal layers. Genuine granules, protruding above the surface of the cornea, are not visible until the cornea is so closely covered with the vesicles that visual acuity is reduced to hand movements before the eye, or to quantitative light perception. Follicles in the pannus, as described by Meyerhof, and the small marginal fossulæ of the resulting limbus may be the only diagnostic signs of a preceding trachoma.

Granules have also been observed in the mucosa of the lachrymal sac.

Healing occurs by gelatinous softening of the granules and the surrounding adenoid tissue. Superficial granules are evacuated outward; the deeper ones lead to connective-tissue formation with subsequent cicatricial changes and shrinking of the conjunctiva.

At the same time the corneal pannus becomes thinner, and the opacity clears up. For a long time afterwards slight mechanical or chemical irritation, such as tobacco smoke, favors the formation of fresh infiltrates in the pannus. During convalescence granules with their concomitant manifestations may also be formed in the previously healthy parts of the conjunctiva.

The *sequelæ* vary. There may be minute, reticular cicatricial striæ, while the conjunctiva of fornix and lids is almost unchanged; or there may be connective-tissue degeneration and considerable attenuation of the entire mucosa (Plate VI, Fig. 1). The latter becomes indurated, loses its natural wrinkling and looks peculiarly smooth and lustrous, with a whitish-blue superficial tint, as if spread with milk. The shrinking process produces a narrowing of the conjunctival sac, the transition folds are shortened or obliterated, so that when the lids are raised or everted, tightly drawn folds become visible in the fornix. At a relatively early stage the tarsal cartilage is affected by the inflammatory process in the same way as the conjunctiva. Later it is distorted into a round or scaphoid shape, or it kinks at a right angle in the subtarsal sulcus, causing the secretory ducts of the meibomian glands to be shortened, partially obliterated, or occluded in the shape of cysts. Shrinking of the conjunctiva further leads to incassation and inflammatory hyperæmia,

entropion, and trichiasis of the palpebral border. All these changes, together with blepharospasm, which is often present, produce a mechanical irritation, causing fresh infiltrations, ulcers, opacities of the cornea, and sometimes keratectasia. If there is grave infiltration of the deeper parts, there will be total or partial keratectasia.

The constantly moist condition of the palpebral border, due to the secretion and consequent wiping, causes cicatricial shortening of the skin of the lower lid. In consequence, aside from entropion of the upper lid, there is often at the inner canthus unilateral or bilateral ectropion of the lower lid, which may be confined to an eversion of the lower punctum lacrimale.

There are also cases which, defying all treatment, lead to incurable blindness, hyaline amyloid degeneration of the tarsus, connective-tissue degeneration of the conjunctiva, obliteration of all glands, with xerotic shrinking of the conjunctival sac up to the cornea, and such a dense opacity of the xerotic cornea that it can hardly be distinguished from the sclera (xerophthalmos).

During examination physicians should always wear protective spectacles.

Diagnosis may be difficult in spite of the most careful inspection.

The following alternatives should be considered: Folliculation, follicular conjunctivitis, specific granular conjunctivitis, incipient conjunctival tuberculosis, ophthalmia nodosa, Parinaud's conjunctivitis, spring catarrh, pneumococcus catarrh of the conjunctiva, and conjunctivitis of the Koch-Weeks bacillus.

Microscopical demonstration of pneumococci or of the Koch-Weeks bacillus in the secretion contra-indicates trachoma, unless there is a mixed infection, which often occurs in trachoma districts.

Differentiation from follicular conjunctivitis is not easy, especially in the initial stage. Follicular catarrh occurs very often in trachoma regions, even in children. There is also a follicular affection resembling trachoma which even the skilled observer may not recognize at once. Non-trachomatous, roundish or oval, so-called lymph-follicles are found not only in the transitional fold of the lower lid, but often, upon thorough eversion of the lids, in the commissures. If they are restricted to the latter parts, it is a symptom against trachoma. The follicles in follicular conjunctivitis are usually arranged in rows (Plate V, Fig. 5), as small, sharply-demarcated, transparent prominences on the surface of the mucosa. Ordinarily they do not encroach upon the tarsal conjunctiva, and they have a yellowish-white or pale red appearance, in contrast to the coarser trachoma granules. These have rather a grayish tint, especially in advanced inflammation. The follicles spring up rapidly and in large numbers, and also disappear rapidly without becoming

gelatinous; the trachoma granules develop slowly, both in size and number. It takes several months for them to become gelatinous, and they are either evacuated by the bursting of the epithelial covering, or neighboring granules coalesce and form larger, gelatinous accumulations.

Neither typical follicular conjunctivitis nor follicular catarrh leads to corneal pannus after prolonged use of atropine or pilocarpine. The former affection is usually confined to the lower transition fold. The secretion is scantier when there is considerable injection and velvety chemosis of the conjunctiva. Follicular conjunctivitis is often accompanied by seborrhoea sicca.

If correctly diagnosed and carefully treated, acute specific granular conjunctivitis (comp. p. 181) leaves no scars in the conjunctiva. This is true in Parinaud's conjunctivitis also (comp. p. 183), with formation of granules. The latter is nearly always unilateral, and sets in acutely along with disturbance of the general condition. Microscopic examination will show the presence of incipient conjunctival tuberculosis, which may simulate trachoma. Here, again, one eye only is usually affected, and there is early tissue degeneration. Ophthalmia nodosa is dealt with under the heading of "Injuries."

Spring catarrh is a complaint of the warm season which causes proliferations of the limbus of the conjunctiva (Plate VI, Fig. 2). The milky appearance of the pavement-like prominences of the upper tarsal mucosa distinguishes it from trachoma. Noteworthy unevenness in the transition fold and eosinophile cells in the conjunctival secretion are absent. Trachoma, on the other hand, is usually located in the adenoid tissue of the conjunctiva, and heals only by the transformation of the affected parts into scar tissue.

The *prognosis* varies according to whether one or both transition folds have been exclusively affected, or whether the tarsobulbar conjunctiva and the cornea are likewise involved. One must also consider whether there are solid or gelatinous granules, or whether the infiltrations have already changed to cicatricial connective tissue. In any case, trachoma in infancy and childhood runs exactly the same course as in adults.

Another factor is the general state of health. This is proved by the differing course of trachoma, of apparently equal gravity in poor districts and in those better situated. One must also decide whether granular conjunctivitis is complicated with oedematous catarrh of the conjunctiva—the "actual bacterial" catarrh, to which school children are principally exposed—or with phlyctenular conjunctivitis and keratitis and other scrofulous changes of the eyes. The method of treatment is also important.

There are more difficulties to be met in seriously scrofulous children, the cornea being often affected, owing to blepharospasm.

Slight relapses often occur. According to Junius, this is explained by the fact that in infantile trachoma the correct treatment is conservative, with the greatest possible limitation of operative shortening of the conjunctiva.

With these restrictions, the prognosis is favorable, provided the affection has been confined to the palpebral conjunctiva and is sharply separated from the healthy parts. It is less favorable if it is less demarcated or the bulbar conjunctiva has been involved. It is less favorable still in the presence of a thin, old pannus, with infiltrations covering the pupillary region, and of corneal opacities.

Treatment.—Statistics show that systematic treatment of trachoma during school age meets with great success. Thus in the county of Königsberg (Prussia), from 1899 to 1905, the number of affected school children decreased from 13.8 to 3.57 per cent.; in the same period from 8.5 to 3 per cent. in the county of Gumbinnen; from 7.71 to 3.64 per cent. in Wartenberg, near Breslau; and from 20 to 4.5 per cent. from 1899 to 1908 in the previously highly-infected district of Allenstein.

The successful treatment of trachoma requires such extensive experience that the school physician as well as the general practitioner will always do well to consult an ophthalmologist. This holds good not only for suspected trachoma, but also in deciding whether children with a slight secretion may be permitted to attend school. Consultation is still more important in fresh trachoma, and in advanced stages where the conjunctiva is already cicatricially contracted, and where other pathologic conditions have developed.

Prophylaxis.—To prevent the infection of healthy children, patients should be immediately isolated, in rooms which are well ventilated day and night. The greatest cleanliness is to be observed, including separate washing utensils, soap, towels, and dishes for the careful cleansing of the hands with oxycyanate of mercury, sublamine (1 : 2000), or sublimate (1 : 5000). These precautions apply also to the physicians and the nursing staff.

The absorbent material used for cleansing the eyes is moistened with 3 per cent. borax and boric acid and immediately burned after use.

Complications are pathological changes of the lachrymal and accessory air passages (hypertrophy, atrophy, ozæna, follicular faucial inflammation, etc.). They should be treated according to indications. Good results have followed the internal use of quinine and arsenic water, like the Duerckheim and Levico waters, in alternate larger and smaller doses. Strengthening diet and correct individualization in hydrotherapeutic measures also promote metabolism. A great deal of time should be spent out of doors and the general conditions improved in every way possible.

The object of local treatment is to effect rapid absorption of the granules without shrinking of the cornea. Persistent blepharospasm or contraction of the lids requires dilatation in the manner explained on p. 101. The conjunctiva should be painted with 2 per cent. silver nitrate, if there is much secretion. The inflammatory symptoms may be mitigated by instillations of zinc, and cooling applications. When they have subsided, the hypertrophic retrotarsal folds are touched with a smooth copper or alum stick, the applications gradually decreasing in intensity. This is followed by massage with a 1-2 per cent. ointment of yellow oxide of mercury or white precipitate ointment, and, according to circumstances, careful touching of the hypertrophic parts of the mucosa and the pannus with plain or concentrated tincture of iodine (concentrated to one-half to one-third of its volume). Intermittent powdering of the conjunctiva with borated tannin (3 : 1) or gallicin also gives good results.

The mechanico-operative treatment consists in massage of the conjunctiva with sublimate (1 : 2000), galvanocautery, puncturing and expressing the granules, and expression by means of roller-forceps or an expressor. A more or less extensive incision of the external canthus (comp. p. 166) may be of service in shortened lid fissures and persistent eversion of the lid, or in defective position of the cilia, which cause continuous corneal irritation.

Excision of the conjunctiva, which, as such, is the most effective remedy, should be resorted to only if a grave corneal complication demands it. The remaining scars are usually smooth and lineal, while those in spontaneous or medicinally effected cures often radiate irregularly.

Infusion of macerated jequirity seeds, or jequiritol-Römer, the dose of which can be more easily regulated, is indicated only in old corneal pannus, which has already partly changed into connective tissue and defies all other treatment. Such treatment only should be carried out when the lachrymal passages are normal, in the absence of epithelial defects, corneal ulcers, and severe conjunctival inflammation. It should be remembered that jequirity causes more or less severe croupous inflammation of the conjunctiva, often accompanied by swelling of the pre-auricular gland, which involves danger to the cornea from necrosis. After instillation, the patient's head is turned outward, and the lachrymal sac is compressed for a while, in order to prevent any part of the fluid entering the excretory lachrymal ducts and causing serious irritation. Such conditions have been repeatedly observed in careless use of jequiritol, and in the presence of dacryocystitis or a tendency thereto.

Goldzieher cured persistent pannus, which was no longer susceptible to any other mode of treatment, by inoculation of gonorrhœal secretion (Piringer). This treatment is by no means devoid of danger, and belongs exclusively to the domain of the specialist.

I agree with Junius in advising the expression of the genuine gelatinous granules, but not others. Painting the transition folds with a 2 per cent. solution of silver nitrate is very efficacious in inflammatory symptoms and copious secretion—conditions prevailing in fresh cases—and at the same time favors healing of progressive corneal ulcers that may be present.

The alum or copper stick is applied to the affected parts of the palpebral conjunctiva with uniformity and great care, the applications being best made in the forenoon. The parts are then sponged with a sterilized cotton pad in order to prevent excessive irritation from the copper salt, which is liberated by the chemical action of the lachrymal fluid. With the patient in the recumbent position cold compresses are applied for ten or fifteen minutes. The application of the copper stick is discontinued if it leads to persistent increase of conjunctival irritation. It is, therefore, contra-indicated in great irritation of the conjunctiva, and particularly in fresh corneal ulcers which are still coated with pus.

If there are no cicatricial contractions of the conjunctiva, the application of the copper stick is continued until the hypertrophic conditions of the conjunctiva disappear. It may, therefore, have to be continued for months or years, with varying assiduity. At first the applications are made daily, later every two or three days, alternating with the milder alum stick. Intercurrent powdering with borated tannin or gallicin is also to be commended.

Purtschler, Sattler, and Zirm recommend soluble copper citrate in the shape of "Cusylol," a chemical composition of copper citrate and sodium borocitrate. According to F. R. v. Arlt, it is but slightly hygroscopic, very easily soluble, and eminently bactericidal, has no caustic effect, and causes but little pain and irritation. Cusylol collyria, containing $\frac{1}{2}$ –1 per cent. soluble copper citrate, are particularly well tolerated in old cicatricial trachoma; those containing 1–2 per cent. are indicated, together with mechanical treatment, in fresh and old trachoma cases without excessive secretion. In addition to applying the solution in the morning and at noon, cusylol ointment may be used late in the evening.

This ointment is prepared with 5–10 per cent. of the following "pulvis cusyloli ad unguentum Arlt":

Cuprum citricum amorph. "Afga".....	100
Cusylol.....	10
Sodium chlorat.....	16
Sodium borocitric "Afga".....	8

and glycerine ointment. It is indicated in cases with slight secretion. The patient, standing before a mirror, applies the ointment to the conjunctival sac with a rounded glass rod, using a bit the size of a pea. The eye is then closed and lightly massaged for about half a minute. Applications are made three times daily.

When the secretion is profuse, v. Arlt powders one to three times daily with itrol-Credé (for eye use), which is kept in blue bottles containing $\frac{1}{2}$ Gm. each. This will soon make the eyes responsive to the copper preparations.

The cusylol dusting powder "*pulvis cusyloli ad inspersionem Arlt*" (copper citrate 5 parts mixed with 80 parts of sterile wheat flour) is intended for the gravest forms of trachoma. This means acute fresh cases, in which strong solutions of silver nitrate would otherwise have been employed.

Further experience is desirable in regard to the copper preparations recommended by Grunert and made with alapurin and white vaseline (Chesebrough), after the method of Schweissinger's eye salve—namely, Zerminol ointment and Zerminol cream. The same may be said in regard to treatment with the carbonic acid snow, which has been successfully employed by Harston in fifty cases of trachoma.

Massage is concentrated upon the region of the upper lid above the superior tarsus, corresponding to the principal seat of the changes.

Tincture of iodine is also used as a collyrium (tr. iod. 1.0, glyc. 15.0); also iodoform, either in substance or as a salve; or pure iodine ($\frac{1}{2}$ –1 per cent.), either dissolved in glycerine or made into a stick of pure iodic acid with very little water. This is used for cauterization every three days. It causes intense pain, which, however, rapidly subsides.

Boric tannin and gallicin powders, in very fine distribution, are sprinkled in a thin layer upon the tarsal conjunctiva, after which the eyes are kept gently closed for one or two minutes. Any excess is rinsed out with tepid water.

The jequirity treatment can be carried out only in a hospital. Any undue effect of this remedy is rapidly neutralized by instilling jequiritol serum. The neutralizing effect is enhanced by subcutaneous injection.

Indications for the various methods of treatment depend upon the pathological picture. In protracted, and especially old, cases operative treatment cannot always be avoided.

The troublesome sensation of continuous dryness in xerophthalmos, which also occurs unilaterally, is relieved by partially suturing the lids, as recommended by Kuhnt. Other methods of treatment have been discussed under the heads of pemphigus, xerosis of the conjunctiva, marantic necrosis of the cornea, and keratitis e lagophthalmos.

9. CONJUNCTIVAL FOLLICULOSIS; FOLLICULAR CATARRH

The occurrence of so-called lymph-follicles, folliculosis conjunctivæ (Plate V, Fig. 5), has been described on p. 173. It is often observed in children in badly-ventilated schoolrooms or institutions. No importance

attaches to it, so long as the surrounding mucosa is only slightly hyperæmic. The follicles will disappear sooner or later without treatment.

In follicular catarrh, however, the conjunctiva is more or less inflamed and chemotic, but the follicles may still be recognized. This is unlike trachomatous infiltration, which soon covers the tarsus and the meibomian glands.

But if the further course must be awaited in order to clear up the nature of the case, the delay is not of great therapeutic importance, since the local treatment which is instituted in violent irritation of the conjunctiva, especially if the follicular catarrh is acute, stormy, and endemic, suffices in incipient trachoma also. Instillation of zinc sulphate ($\frac{1}{4}$ – $\frac{1}{2}$ per cent.) or of 2 per cent. cusuylol, painting with 1 per cent. silver nitrate, cooling applications to the eye, give good results. Straining the eyes is to be avoided. There should be good illumination and ventilation of the dwelling and sleeping rooms day and night, and much exercise in the open air. In epidemics schools and institutions may have to be temporarily closed. If the conjunctiva is otherwise healthy, instillations of sodium biborate 0.2 : 10.0 may be made as a palliation, since these follicles will gradually disappear without leaving a trace.

The presence of a profuse number of follicles complicates the treatment of chronic catarrh of the conjunctiva.

The use of atropine or pilocarpine is discontinued when follicular catarrh sets in; other mydriatics or miotics may, however, be substituted.

10. SYPHILITIC AFFECTIONS OF THE CONJUNCTIVA

Independent initial sclerosis occurs very rarely in the conjunctiva. The infection takes place most frequently in the places of most easy access, that is to say, the mucosa of the lower lid and the inferior conjunctiva of the fornix. The orbital conjunctiva, plica semilunaris, and caruncula are less often attacked, and the mucosa of the upper lid very rarely. The infection is mostly communicated to the eye by contact. In one case, a trachomatous conjunctiva was actually treated with a brush that had been previously used for cauterizing a syphilitic case.

Differential Diagnosis.—In addition to the history, the Wassermann, Bruck, and Neisser tests, such general essential points as skin exanthema, mucous plaques in the mouth, condyloma of the anus (although these do not always become apparent until a later stage), and indolent swelling of the neighboring lymph-glands, the clinical picture will distinguish the condition from a broad pustule, an episcleritic node, or slowly developing tumor of the conjunctiva. It shows an absence of increased secretion, and in the centre of the surface of the indurated and infiltrated growth there is either a yellowish spot or a very sensitive ulcer with a discolored gray or yellowish, lardaceous base and with serrated, precipitous, and

infiltrated borders. There is considerable œdema and catarrhal turgescence of the surrounding conjunctiva, and the skin of the swollen lid over the tumor is inflamed. The palpebral and orbital conjunctivæ are sometimes disintegrated at opposite points, where the ulcerated surfaces touch when the lids are closed. In a five-year-old girl, with eczema impetiginosum necroticum of the lids, the primary affection of the lower lid had apparently produced a new pathological focus at the opposite point on the bulbar conjunctiva. In chancre of the upper part of the palpebral conjunctiva there is redness and perhaps ptosis, of a more or less pronounced degree. During and after the cicatricial stage, the condition may be confused with burns, diphtherial infiltration, conjunctival tuberculosis, and pemphigus. According to its location and extent, cicatrization may cause shortening of the mucosa or symblepharon of varying degrees, along with indurations which are visible for a considerable time.

Antisyphilitic treatment in dubious cases may decide the question, although this is sometimes a failure (Peppmüller).

Treatment.—In the first place, constitutional treatment is instituted. Locally, inspergation of calomel or iodoform is used, intermittent light massage of the conjunctiva with 0.5–1 per cent. salve of yellow oxide of mercury, and cool borated applications.

Obstinate CONJUNCTIVAL CATARRH with chemosis of the bulbar conjunctiva is often accompanied by severe headache at night and hyperæmia of the lid and conjunctiva. These are sometimes evidences of constitutional syphilis, and sometimes are precursors of syphilitic iritis, or papulous cutaneous syphilis. Hyperæmia of the conjunctiva occurs also in children with hereditary syphilis.

Constitutional syphilis is often accompanied by hyperæmia and obstinate catarrh of the conjunctiva, marginal phlyctenæ and multiple hæmorrhagias of the retrotarsal conjunctiva. In other cases the conjunctiva is unusually pale; in others, again, yellow, as, for instance, in the icterus of the early stage of syphilis.

Local treatment is instituted when needed. See p. 130.

In recent syphilitic cases swelling of the lymph-follicles in the adenoid layer between the epithelium and tarsus, usually near the fusion with the lower lid, will occur accompanied by considerable turgescence of the pre-auricular and buccal lymph-glands. There are slight or no changes of the conjunctiva. These follicles disappear under specific treatment without local interference. Numerous pale yellow granulations, resembling trachoma granules, have been observed in the diffusely infiltrated mucosa. The latter has a colloid, lustrous, anæmic, and in places ecchymotic appearance. The lids, notably the lower one, are thickened throughout. This very rare form, designated as “acute specific

granular conjunctivitis," is recognized by the turgescence of the lymph-glands, especially in the anterior and posterior parts of the neck, by simultaneous or preceding syphilitic manifestations at other parts of the body (cornea, iris, etc., so far as eyes are concerned), and the failure of the local remedies which are effective in granular conjunctivitis. These remedies, in fact, may cause an exacerbation. On the other hand, systematic treatment with mercury and potassium iodide will effect a complete cure.

The same may be said of papulous conjunctival syphilides, as was observed by France and others in infantile hereditary syphilis. The papules of the MUCOSA and CONDYLOMATA of the conjunctiva are either flat or raised, in size from a pin head to a pea, and may be mistaken for new growths. But in nearly all cases there are also papules upon other parts of the body. They are for the most part of the same consistency as the typical papules of the mucosa, and evince a predilection for the plica semilunaris and the caruncle, where one or more of them will be found. In some cases a papule of the palpebral conjunctiva will be followed by a similar one at the opposite point on the bulbar conjunctiva. They show no particular tendency to ulcerous disintegration, unlike the papules of the palpebral border, which are subjected to maceration through lachrymal fluid and conjunctival secretion, and to mechanical injuries from rubbing the eye. Their local antisymphilitic treatment is therefore unnecessary, as they disappear under constitutional treatment, leaving either no trace at all, or only a pigmented brownish macula or a slight vascular injection.

The papules appear on the hyperæmic and often chemotic and coated conjunctiva as a flat spot or as oval, coarse interstratifications of grayish-red or reddish-yellow coloration, or as prominences which resemble large phlyctenæ or fibromata. They differ from phlyctenæ by their gradual growth, and from fibroma by an early arrest of development.

The local treatment is symptomatic (comp. p. 130). If a papule is located near the corneal margin, the same local treatment may be necessary as for marginal ulcers of the cornea.

GUMMA of the orbital conjunctiva has been seen in isolated cases of hereditary syphilis, and is found almost exclusively in the region of insertion of the external rectus or in the sclera. It may encroach upon the corneal limbus and the cornea itself. It begins as a firm, not sharply-demarcated tumor of yellowish appearance, which is immovable or slightly movable on the surface of the sclera. It is accompanied by evidences of grave iridocyclitis, such as synechiæ, vitreous opacities, etc., and may either disappear, leaving a pigmented spot on the sclera, or disintegrate, forming an ulcer with an indurated, infiltrated yellow base and eroded borders. In primary involvement of the sclera the process

may lead to perforation and disintegration of the entire orbit. Gumma of the palpebral conjunctiva resembles an infiltrated meibomian gland which has perforated toward the conjunctiva. It is accompanied by a certain degree of diffuse swelling of the conjunctiva and the entire lid. When cured, either a scar or a pseudopterygium of the conjunctiva is to be expected. If located in the submucosa of the lid, there may be destruction of the tarsus, with shortening and eversion of the lid. The development of gumma of the conjunctiva is sometimes attended with violent pain and irritative manifestations.

The *diagnosis* is suggested by the appearance of simultaneous gummata in the iris and corpus ciliare, as well as by homogeneous or heterogeneous syphilis at other parts of the body. In order to avoid mistaking gumma for other malignant new formations, which have often been repeatedly and uselessly operated upon, it is important to note that the latter usually form warts on the conjunctiva, while gummatous ulcers protrude but slightly over the level of the conjunctiva and form excavations by ulcerous disintegration.

Local Treatment.—Comp. p. 130. Impending perforation of the sclera may, under certain circumstances, be arrested by a duplex bandage. If phthisis bulbi develops, enucleation or evisceration may become necessary.

Exanthematous conjunctivitis, due to hereditary syphilis in premature infants, which Hirschberg observed on the seventeenth day after birth, was promptly improved by sublimate baths.

Syphilitic pemphigus has been repeatedly observed in the first year of life. It causes shrinking of the conjunctiva with cicatrization. Constitutional treatment has arrested the local process.

II. TUBERCULOSIS OF THE CONJUNCTIVA

Tuberculosis of the conjunctiva is nearly always unilateral and usually occurs between the tenth and thirtieth years. It is caused either by ectogenous infection (foreign bodies infected with tuberculosis or tuberculous sputum) or it is of hæmatogenous origin. This latter appears as trachoma-like miliary tubercles situated both at the transition fold and in the bulbar conjunctiva itself. Lupous tuberculous foci of the cutaneous surface of the lid or at the nasal mucosa may spread through the lachrymal vessels to the conjunctiva. The reverse may also happen.

The process usually sets in at the conjunctiva of the upper lid, with a preference for the subtarsal sulcus, the principal seat of stationary "foreign bodies." It then spreads, in the shape of a painless ulcer characterized by a lardaceous, dirty yellowish-red base, uneven edges, cockscomb proliferations, and small, yellowish-gray nodules, sometimes

resembling lymph-follicles. There are also considerable turgescence and incassation of the affected lid, principally of the intermarginal part, and abnormal mucous secretion which, in the course of months or years, spreads to both the deep and superficial adjacent parts. Neglected endogenous miliary tubercles may resemble trachomatous follicles, and are often accompanied by catarrhal chemosis of the conjunctiva, but they gradually disappear under galvanocautery without leaving noticeable scars, while such a favorable termination is extremely rare in discrete tuberculous proliferations.

There is no doubt that the process may sometimes be arrested. In most cases, however, the ulcer advances toward the bulbar conjunctiva and cornea, often with the formation of a pannus of varying density, which has a predilection for the upper marginal section. If the ulcer erodes its way toward the palpebral skin, there will be defects of the lids with symblepharon, destruction of the cornea, or phthisis bulbi. The conjunctiva may also be secondarily affected by a tuberculosis which was primarily intra-ocular or scleral. Swelling of the equilateral preauricular, maxillary, and cervical lymph-glands occurs either at an early or late stage, or it may be hardly noticeable. Death from general tuberculosis has occurred.

Differential Diagnosis.—Syphilitic ulcers, glanders, pemphigus, chalazion, circumscribed necrotic staphylococcus diphtheria, variolar pustules, vaccine ulcers, disintegrating epithelioma of the conjunctiva, must all be considered. The diagnosis is assured if any of the established tests show the presence of tuberculosis.

The prognosis in chronic conjunctival tuberculosis is, generally speaking, unfavorable, as medical aid is usually not summoned until there has been considerable local devastation. This is especially true if the patient has other centres of tuberculous infection. These complications are an important factor in the nodular infiltrates of the bulbar conjunctiva, because these eczematous pustular efflorescences may spread to the inner surface of the sclera and undergo ulcerous disintegration.

Treatment.—Galvanocautery is indicated if the tuberculous focus is so circumscribed that it can be completely removed; otherwise, undiluted or 50 per cent. lactic acid may be applied to the affected places, or the Finsen light illumination after Lundsgaard's approved technic. Tuberculin has also been favorably reported upon. Long-continued application of iodoform or airol, either as a finely-distributed powder or as 10–20 per cent. salve, to the conjunctiva, has achieved good results.

Kuhnt observed a permanent cure of extensive tuberculosis of the conjunctiva and cornea, thanks to the occurrence of facial erysipelas.

The affection described as Parinaud's "*conjunctivite infectieuse d'origine animale*" is probably a BOVINE CONJUNCTIVAL TUBERCULOSIS. It

is usually unilateral, and most frequently attacks butchers, cattle dealers, cowboys, stablemen and their families. The pre-auricular and sub-maxillary glands are swollen and suppurating, and simultaneously there develop in the conjunctiva of the lids near the transition folds large and small granular, papillomatous, cockscomb proliferations, and small yellow nodules resembling trachoma granules and tubercles. They disappear without leaving a trace after having run a protracted course covering several months. Chills and fever do not often occur.

Corneal complications are rare. In one case, with perforation of a small ulcer, streptococci were cultivated from the conjunctival sac.

Treatment.—Incision of the infiltrated glands may become necessary, but as a rule it suffices to protect the eye from further injury, and to apply non-irritative dressings, such as 3 per cent. boric acid.

12. LEPROUS AFFECTIONS OF THE CONJUNCTIVA

These affections are usually secondary. In the anæsthetic form there is either a highly anæmic or a hyperæmic catarrhal swelling of the conjunctiva. In ectropion and lagophthalmos there will be increased lachrymation or blennorrhagic traumatic conjunctivitis, which in time leads to atrophy of the conjunctiva, to pale, lardaceous, lustrous incrassation of the lid fissure part of the bulbar conjunctiva, to pterygium, conjunctival xerosis, and symblepharon.

In the tuberos form, the tarsal conjunctiva is usually diffusely enlarged or impaired by scars due to ulcerous disintegration of flat, fungiform nodules. Nodular infiltrates appear in the bulbar conjunctiva and episclera in close propinquity to the corneal margin, less often in the sclera. They are glassy prominences, at first white, yellowish-white, or reddish-white. Later they become yellowish, with a lardaceous lustre and a precipitous slope toward the adjacent parts of the opaque cornea. They become flattened toward the sclera, and often encroach upon the cornea, the periphery of the iris, and the ciliary body.

For *treatment*, see p. 130. Terson reports a case in which leprous nodules of the episclera and the palpebral borders disappeared when facial erysipelas set in.

Ecklund states that the affection is often communicated to the conjunctiva by the indiscriminate use of towels.

13. SPRING CATARRH (SAEMISCH, PLATE VI, FIG. 2)

HYPERTROPHIE PÉRIKÉRATIQUE DE LA CONJUNCTIVE (DESMARRES)

Spring catarrh occurs almost exclusively in childhood and in the male sex. The external appearance of the patient is flabby and sleepy, due to pseudoptosis of varying degree. A proliferation of the scleral conjunctiva develops at the corneal limbus, with symptoms of a con-

junctival catarrh, which, however, is often devoid of importance. The appearance of the proliferation varies. Sometimes it is a diffuse grayish turgescence of the enlarged limbus, with small, transparent, whitish, warty eminences, sharply demarcated against the cornea or overtopping it. Sometimes it is a pale, dirty gray, yellow, reddish-gray or brownish-gray, gelatinous, warty eminence, which looks like wax and spreads toward the conjunctiva and cornea. Here it may connect with a circumscribed opacity of the adjacent corneal section resembling the arcus senilis. The inferior tarsal conjunctiva is generally pale, dull, somewhat thick and opaque, "as if covered with a thin layer of milk," or it is pervaded by minute, whitish, retiform cicatricial cords. This is often accompanied at the upper lid—less often at the lower—by papillary proliferations of the tarsal conjunctiva, in the shape of hard, partly fungiform, dull white or grayish-pink, flat, dry eminences disintegrated at the surface. It resembles an "irregular street pavement."

Differential Diagnosis.—As to trachoma, comp. p. 174. The initial stage of the orbital changes is often mistaken for eczematous conjunctivitis.

The subjective complaints are lassitude and heaviness of the lids. They occur almost regularly as the warm weather sets in, subside in the summer during even a short cool spell, and completely disappear in the fall and winter. The objective changes, as described, persist unchanged for years, but gradually disappear almost entirely. Visible traces, however, often persist, such as a narrowing of the corneal area by a grayish marginal opacity—which only rarely resembles the arcus senilis—slight ptosis, and milky opacity of the palpebral conjunctiva.

The general examination has often shown anæmia. The secretion and the conjunctiva reveal abundant eosinophile cells. Schleich and v. Michel observed universal polyadenitis. The hæmoglobin count was reduced in a few cases, with leucocytosis of varying extent. Kreibich attributes the affection to the chemical influence of the sun's rays. This is supported by the investigations of Reis, who found changes in the subtarsal vessels which closely resembled degeneration of the endothelia of the intima produced by Röntgen rays, radium, or high-frequency currents. I observed a fourteen-year-old boy in the high school at Bamberg whose pathological manifestations disappeared spontaneously during the summer, which he spent in the forest of Franconia.

Treatment.—A change of residence may be necessary, woods in a medium altitude being preferable. Strengthening diet is indicated and improvement of the metabolism, with administration of the following remedies, to be prescribed alternately: Fowler's solution and syrup of the iodide of iron. Locally: Yellow or smoked protective spectacles. Palliative: Ice compresses, instillations of five drops of acetic acid

(diluted in 10.0 aq. dest.); of tinct. opii crocat. with suprarenin āā; of holocaine 0.01, alypin 0.2, adrenalin (1.0 : 1000.0), 0.1 aq. dest. 15.0; or inspergation of xeroform. Massaging the conjunctiva may have a favorable effect.

Von Michel advises extensive and energetic removal of the firmly adherent proliferations with the sharp spoon. "Very often it requires the aid of scissors to remove the affected tarsal tissue."

A. and J. Lawson report the "veritably specific effect of radium," leaving "the scars smooth, tender and pale." Further experience should be awaited regarding this treatment, as well as the prophylactic effect of zeozon instillation.

14. LYMPHOMA OF THE CONJUNCTIVA

In cases reported by Greeff and Goldzieher, the transition folds and the palpebral conjunctiva of the affected eye were so œdematous that when the lids were everted the latter presented in the shape of ridges. The hyperæmic mucosa was covered with large numbers of "follicular" vegetations, from miliary to lentil and bean size, resembling caruncles, which protruded at the conjunctiva of the superior fornix. Goldzieher found also rather hard, lymphomatous bundles on the same side of the neck, and Greeff moderate swelling of the equilateral pre-auricular gland.

Treatment.—In Greeff's case the symptoms gradually subsided under the influence of cold applications, instillation of $\frac{1}{2}$ per cent. copper sulphate, and hygienic measures. Goldzieher removed the large nodules with scissors, and cauterized the bleeding places with the galvanic current. The latter was also used to puncture the smaller follicles. He then prescribed daily irrigations of the conjunctiva with sublimate solution, and internally arsenic mineral waters. This treatment caused the cervical lymphomata to subside rapidly.

15. EPITHELIAL XEROSIS OF THE CONJUNCTIVA

With the exception of parenchymatous xerosis—desiccation of the conjunctiva and cornea (a sequel to granular conjunctivitis)—or xerosis in pemphigus, after diphtherial conjunctivitis, in paralysis of the facial nerve, and in pronounced exophthalmos and ectropion, it may be said that benign epithelial xerosis never leads to serious affections of the cornea. It is almost without exception bilateral. The exposed part of the bulbar conjunctiva is slightly thickened in the shape of an isosceles triangle, with the base at the corneal margin. In appearance it is dull, without lustre, as if covered with dried foam or whitish, lardaceous, shining deposits (so-called Bitôt's spots). They have been observed in anæmic cases, both scrofular and tubercular, and are endemic in orphan

asylums; but they are also found in well-nourished and usually healthy children. There is often decreased lachrymal secretion and hæmeralopia (torpor retinæ), caused by insufficient nutrition of the retina.

The *treatment* is principally causal. Locally, it consists in improving the dry condition of the eye, and inciting lachrymal secretion by moist heat and protective bandages. For night-blindness dark protective spectacles should be used. As to malignant epithelial xerosis, see p. 203.

16. PSEUDOPTERYGIUM

Pterygoid or pseudopterygium usually originates by contact of the chemotic, infiltrated, conjunctival limbus with a large gonorrhœal or diphtherial corneal ulcer, or in the cicatricial stage of granular conjunctivitis. It may also be produced by burns and erosions of the bulbar conjunctiva and cornea, after operative removal of neoplasms at the corneal margin. It is less frequently caused by marginal affections of the cornea, as, for instance, rodent ulcer. Its shape, form, and extent vary considerably. It is sometimes surrounded by an extensive leucomatous corneal opacity, and, unlike true pterygium, is not confined to the nasal or temporal side of the cornea, but is also found at the upper and lower corneal margin; it does not grow larger, and disappears in a short time. In the pseudo form there is often a symblepharon due to erosions or burns. Furthermore, it is adherent to the cornea by the apex only, and can be easily brought into the correct position by operation.

If, however, the conjunctiva is dragged into a torpid, superficial marginal ulcer of the cornea, it adheres to the base in its entire extent. It may then, like true pterygium, advance toward the centre of the cornea.

Treatment.—The operative removal of the disturbances, which manifest themselves in the form of mechanical amblyopia, restricted motility of the eye, impeded lachrymal absorption, and irritative conditions, should be referred to the ophthalmic surgeon.

17. CIRCULATORY DISTURBANCES AND HEMORRHAGES

In all forms of chronic anæmia there is pallor or slight icterus of the conjunctiva, and venous hyperæmia of the conjunctiva. With it there are sensations of "dryness"—"sand grains in the eye"—heaviness and ocular fatigue, which symptoms are more prominent in the morning.

The *treatment* is causal and local: Instillation of tinct. opium 1-3 : 10-15 distilled water. More energetic remedies are not borne well.

Spontaneous extravasations of blood under and into the conjunctiva, the semilunar fold, and caruncle occur in varying color, thickness, and extent in all conjunctival inflammations. They are more often seen in the bulbar portion and are especially prominent in the upper half of the bulbar in pneumococcus infections. Subconjunctival hemorrhages are common

in scorbutus, leukæmia and other general diseases, especially pertussis (Plate III, Fig. 4), as well as in contusions and head injuries of all kinds.

These hemorrhages usually disappear spontaneously, but hemorrhages of the free surface of the conjunctiva, in hæmophiliacs, are not without danger, as many cases have bled to death. In a squint operation on such a subject it became necessary to resort to transfusion.

Treatment.—In scorbutus and Barlow's disease—the scorbutus of infancy—rational diet is of the greatest importance, as explained on p. 141. In extensive hemorrhages occurring in new formations and diffuse inflammations pressure bandages may become necessary. In uncontrollable hemorrhage the gentle hæmostatic remedies, such as suprarenin or alum, are to be tried first. The lids should be closed by a continuous suture, and subcutaneous or intravenous infusion of salt solution tried.

Extravasation of Serum under the Conjunctiva.—Chemosis is caused by plethora of the conjunctival vessels and the pericorneal subconjunctival vessel, or by stagnation of the blood and lymph in the conjunctiva, owing to dacryocystitis, periostitis of the orbital margin, or hordeolum near the outer canthus. These conditions, hyperæmia of the conjunctiva and swelling catarrh of long standing, usually give rise to a mild, more or less transparent, cedema. On the other hand, in acute gonorrhœal, trachomatous, croupous, or diphtherial conjunctivitis, in acute inflammation of the uveal tract, cyclitis, acute glaucoma, panophthalmitis and orbital phlegmons, the secretion under the bulbar conjunctiva may be so viscid or fibrinous that the raised part has a gelatinous, yellowish opaque appearance, towering over the orbital margin like a rampart.

Serous chemosis is due also to occlusion of the afferent lymph-tracts, as in suppuration of the cervical lymph-glands; or it may be due to blood extravasations under the conjunctiva, owing to the spreading of inflammatory products in the orbit. These forms of chemosis are often accompanied by cedema of the lids, especially in acute attacks. In severe inflammations of the conjunctiva or uveal tract the chemosis may interfere with the closing of the lids. In inflammations of the conjunctiva and cornea, attended with pronounced photophobia, the upper and lower palpebral conjunctiva may bulge out (ectropium spasticum), and if it cannot be reduced, several scarifications of the conjunctiva may become necessary.

The hyperæmic chemosis of Bright's disease recurs from time to time, with rapid onset and rapid disappearance.

In filtration chemosis the aqueous humor infiltrates under the conjunctiva, owing to marginal corneal fistulæ, the defective closure of a trauma in the wall of the anterior chamber, or to impaction of part of the iris in the traumatic canal (cystoid cicatrization).

18. NEW GROWTHS OF THE CONJUNCTIVA

Congenital new growths are dealt with on p. 11. Those acquired in infancy and childhood affect principally the bulbar conjunctiva and fornices. They include lymph-cysts, serous epithelial cysts, cysts due to ligation or fold-formation of the conjunctiva following chronic blennorrhœa and granular conjunctivitis, subconjunctival serous retention cysts of the glands of Krause, proliferation cysts of spring catarrh, hydatoid cysts and traumatic cysts, and cysts due to destruction of the bulbar capsule.

Treatment.—If cysts produce irritation or other inconvenience, their tips are removed with a small, sharp hook, and the walls scraped with the sharp spoon or carefully cauterized with the galvanic current or silver nitrochlorate.

Hæmangioma cavernosum is characterized by a dark red or blue-red swelling. It occurs in the bulbar conjunctiva and is often accompanied by vascular affections in other parts of the body due to congenital telangiectasia.

It may also originate from an angioma of the sclera, an angiofibroma of an external ocular muscle; or from an injury, like that of the subcutaneous vessels from application of forceps. It may rapidly spread over the entire bulbar conjunctiva. Angioma occurs most frequently near the inner canthus and the neighboring parts of the bulbar conjunctiva. It may spread from the external surface of the lid to the palpebral conjunctiva.

Treatment.—If possible at all, excision of the angioma is preceded by ligation, and followed by careful cauterization with silver nitrate and tincture ferric chloride in order to prevent recurrence.

Electrolysis has been used with success. Hæmangioma requires removal, if it interferes with lachrymal absorption, gives rise to inflammatory processes in the conjunctiva, impedes the movements of the eye, or endangers the cornea. Enucleation of the affected eye, with extirpation of the new growths surrounding the cornea and of a few foci in Tenon's capsule and in the cellular fat tissue, has been necessary only in a single case, where the exceedingly rapid development of the angioma could not be arrested even by electrolysis.

If, as is rarely the case, a closed, cystic, cavernous lymphangioma develops in the conjunctiva it should be removed.

Papilloma of the conjunctiva has either a slight or intensely red tint. It is of irregular formation, and has an uneven, strawberry-like or disintegrated villous appearance. It is more often multiple than solitary, and has a predilection for the region of the inner canthus and the corneal limbus. Papilloma should be removed as early as possible and, owing to their great tendency to recrudescence, the wound should

be cauterized. This procedure applies also to primary sarcoma of the conjunctiva and epibulbar carcinoma. Both have a great tendency to reoccur. They are most often found in the exceedingly vascular limbus of the conjunctiva. Metastases occur less often. Sarcoma may occasionally develop from a pigmented nævus, often as the effect of a trauma, but it very rarely penetrates into the true corneal tissue, sclera, or the interior of the eye. Carcinoma, on the other hand, often spreads over the cornea and sclera, and even penetrates from the limbus into the interior of the eye, along the lymph-sheaths of the anterior ciliary arteries.

Treatment.—In sarcoma of the palpebral conjunctiva a thorough and early removal is usually sufficient. In epibulbar sarcoma enucleation of the eye, removal of the orbital tissue, and exenteration of the orbit may be necessary, in case the bulbar adnexa and the orbital tissue are involved. This may happen if the other eye is still unaffected, and if the sarcoma again and again recurs and permanent control is uncertain. In epibulbar sarcoma Saemisch advises locating the base first, so as to prevent hemorrhage. In epibulbar carcinoma the defect after operation has often been successfully covered by drawing the adjacent conjunctiva over it.

Of independent affections of the semilunar fold and the caruncle, swelling and hypertrophy due to hemorrhage into the tissue of the semilunar fold have been observed in infancy. So have lymphoma and melanosarcoma.

For ophthalmia nodosa, injuries of the conjunctiva and their sequelæ (symblepharon), see "Injuries."

VII. AFFECTIONS OF THE CORNEA

INSUFFICIENT general nutrition, as well as impaired nutrition of those parts which provide the vascular supply of the corneal margin, exerts an injurious influence upon the cornea. Elevation and maceration of the epithelium—as in stagnation of pathological secretion in the conjunctival sac—will allow injurious substances to exert a detrimental effect upon the corneal parenchyma. This is also favored by a decrease in normal moisture of the anterior segment of the globe, especially as that part of the cornea is normally exposed to increased evaporation. Infections of the intermarginal palpebral fringe, and of the limbus of the bulbar and scleral conjunctivæ, spread by continuity to the cornea. The marginal vascular network, which consists of loops and capillaries, may be responsible for simple as well as septic emboli. The close interrelation between these vessels and those of the conjunctiva, sclera, iris, and ciliary body may, by metastatic embolism and thrombosis, disturb the nutrition of the cornea, and so favor the development of ectogenous infections.

For the same reason, deep corneal changes, including septic ones, often by reaction, cause inflammatory changes in the cornea, sclera, and anterior part of the uveal tract.

Diagnosis.—The following conditions are important: Microcornea, corneal irregularities from old scars, inflammations and extensive opacities; keratoglobus, keratoconus, changes following interstitial keratitis, pannus and excessive cicatricial tissue.

Surface changes are recognized in daylight by the window reflex, or in artificial light by oblique illumination on the anterior surface of the cornea. They are still better recognized with Placido's keratoscope, a metal disc with concentric white and black circles. A substitute may be made with white card-board.

Changes in the lustre and evenness of the corneal surface produce indistinctness and distortion of these pictures, which, under normal conditions, are perfectly sharp and clear. If there is lack of moisture on the surface they appear dull and blurred. In recent parenchymatous inflammation, or in the corneal œdema due to a rise of intra-ocular tension, the surface appears slightly punctated or "breathed on." The reflected picture is like an illuminated surface, if the cornea is dull and dry. Torn, distorted, mixed, and indistinct reflexes are caused by any inflammation of the corneal surface, involving loss of or damage to the epithelium, to new growths or scars, provided there has been loss of tissue in the deeper layers of the cornea.

The cornea is often more or less opaque. The opacity may be superficial, or deep, partial, total, transparent, dense, isolated, in groups or confluent. The design, shape, color, and extent of the opacity may likewise vary, as may the consistency of the surface previous to the opacity. Fresh (inflammatory) opacities show a dull, stippled surface; old (cicatricial) ones are usually shining. The whitish-gray opacity of old age in the marginal zone of the cornea, due to fatty, granular deposits in the lamellæ, may occur in childhood. These deposits are distinguished from the scars of marginal ulcers by their regularity of shape and sharp demarcation toward the limbus. They do not exhibit new blood-vessels.

The layer harboring the opacity can be recognized with a loupe by its parallax displacement. It becomes even more distinct on powdering the cornea with a little calomel.

The origin of opacities varies. In the epithelium they are due to thickening, œdema, or necrosis. The opacity of fascicular keratitis, which is located between the epithelium and the anterior elastic membrane, is due to deposits of lime, hyaline, and fat. In pannus it is caused by deposits from exuding cells and new formation of tissue. In the parenchyma it is due to œdema, hemorrhages, accumulations from exuding cells, necrosis of corneal corpuscles (infiltration), newly-formed connective tissue (scars, callosities), and tumor cells.

The posterior elastic membrane and its epithelium are folded and creased (striated keratitis), and there are ligamental opacities in the deep parts of the corneal substance, caused by tears in the posterior elastic membrane. In infantile glaucoma there are also punctiform, grayish, yellowish-gray, or yellowish-brown deposits on the lower and middle parts of the posterior corneal surface. They are sharply demarcated by exuding cells, fibrinous, coagulating and pigment cells, and are thereby distinguished from the small punctiform infiltrates located in the posterior corneal layer.

Foreign bodies and incrustations of lead, silver, mercury, pigment, etc., may be found in the various corneal layers.

The sensitiveness of the cornea, which may be impaired in herpes, paralysis of the trigeminus, and glaucoma, is tested with a fine cotton tip. Should there be doubts as to whether the dull surface is due to parenchymatous keratitis or acute glaucoma, tonometric examination will decide the question. An epithelial defect of the cornea stains green, upon instillation of 2 per cent. fluorescein. A. v. Reuss uses fluorescein and methylene blue, the latter staining only the corneal substance, so that the blue cornea can be seen within a green areola.

The location of new vessel formation in the cornea is of importance for differential diagnosis. Thus the fine vascular bands (Plate VI, Fig.

6) or pannus (Plate VII, Fig. 2) can be seen as red striæ, running superficially over the limbus, either parallel or converging toward the centre of the cornea, and fusing with the conjunctiva or episclera. In parenchymatous keratitis the vessels often converge toward the limbus, where they terminate and can be followed with the loupe (Plate VII, Fig. 7). Encroachment of the net of marginal loops in the shape of sectors, or in a circle beyond the corneal margin, occurs in superficial and deep affections of the cornea.

Large perforating injuries may even cause inversion or eversion of the corneal wound.

Adhesion of the cornea to the bulbar conjunctiva (pseudopterygium) or to the lids, the conjunctiva of the fornices and of the lids, can easily be recognized.

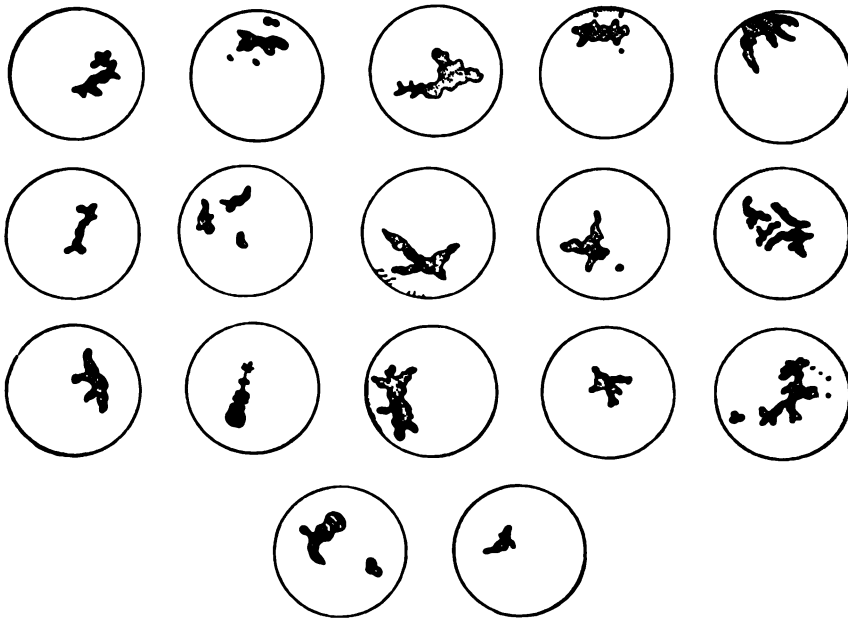
For the detection of the more minute changes in the cornea and the anterior section of the globe, examination with magnification is essential. The Westin-Zehender loupe and the Czapski-Zeiss binocular microscope are both good. The latter has been modified by Amberg for the determination of the depth of the anterior chamber and the other optic constants. Illumination of the sclera with reflected light serves to demonstrate minute corneal opacities, lenticular changes, and especially atrophy of the iris.

Another important question is whether there is an acute conjunctivitis or whether the conjunctival irritation is secondary to a serious corneal affection. This is especially pertinent in the initial stages, where the exact form of the inflammatory reaction should be diagnosed. Pericorneal and ciliary injections, which vary considerably in degree, have a pale violet appearance, distinguishing them from the injection in acute affections of the conjunctiva and lids, which is scarlet or brick-red. They are further differentiated by the fact that the injected ciliary vessels are not clearly outlined, especially when the bulbar conjunctiva shows serous infiltration. Ciliary injection is therefore more diffuse. The plethoric vessels of the bulbar conjunctiva are movable, the deeper scleral and episcleral vessels are not. The ciliary vessels may be emptied by digital pressure, so that for the time being they completely disappear; the dark and larger vessels of the conjunctiva cannot be so easily emptied. Another point is that conjunctival injection is most pronounced in the fornices, and decreases in intensity toward the cornea. In severe inflammatory processes of the cornea, sclera, and the anterior uveal region, both forms of injection may occur together, owing to numerous anastomoses in these regions. For example, in grave inflammations of the conjunctiva and anterior section of the globe the episcleral and conjunctival networks are so congested that an exact differentiation is impossible.

In glaucoma, especially the chronic form, the dilated episcleral ciliary veins are often of a bluish translucence.

An early recognition of the corneal affection is important for correct treatment. The fact can, therefore, not be sufficiently emphasized that repeated and even daily examination is of the greatest importance. This is true in variola, and even in the convalescent stages of infectious diseases. Following tuberculosis and other acute febrile diseases grave eczematous affections have often been observed.

FIG. 28.



Various types of herpes corneae. (After Hagnauer.)

1. HERPES OF THE CORNEA

This disease does not occur as often in children as in adults. It is more frequently associated with influenza, febrile bronchitis, and acute pneumonia than with typhoid fever, pertussis and intermittent fever. It is nearly always unilateral and accompanied by symmetrical herpetic eruptions of the face. Recovery takes place in from two to four or more weeks, and, provided treatment has been correct, without leaving any pronounced trace. Otherwise a corneal opacity of varying degree may persist, perhaps accompanied by posterior synechiæ. There is considerable lachrymation and stabbing, burning pains, together with the sensation of a foreign body in the eye, violent photophobia and redness of the conjunctiva. There may also be blepharospasm, ciliary neuralgia and reduction of the intra-ocular tension. Small vesicles in

varying numbers, from pin-head size to a miliary seed, containing a limpid or slightly turbid fluid, appear anywhere on the corneal surface. The vesicles vary in shape, sometimes forming a bifurcated line, a clover leaf, or rosary figure (Fig. 28).

The epithelial covering is often so rapidly desquamated that the cornea seems to be transparent, enabling the physician to observe a delicately opaque, irregular, zigzag, circumscribed linear marking or stellate, small ulcers, due to repeated new crops of herpes vesicles (*keratitis dendritica*, *ramiformis*, or *stellata*). In the first stage isolated defects may coalesce, or, less often, cause a suppurative corneal infiltra-

FIG. 29.



Illuminating lens. (After Kehr.)

tion accompanied by iridocyclitis and hypopyon. Swelling and hemorrhage of the iris may accompany the disease (Gilbert). Shreds of the vesicular covering may hang down from the invaded areas in the shape of whitish threads. The cornea surrounding the ulcers may have a foggy or light gray appearance or present opaque striations. Unlike herpes zoster ophthalmicus, corneal anæsthesia is confined to areas denuded of epithelium. Pronounced pericorneal injection is the exception. During convalescence, glaucoma may appear.

Even with lateral illumination (Fig. 29), which is indispensable in examination, all these changes in the anterior segment of the globe cannot always be easily recognized.

In doubtful cases a 2 per cent. solution of fluorescin is instilled, the lids are closed for about thirty seconds, and the excess removed with a

4 per cent. solution of sodium carbonate. The epithelial defects of the cornea are then stained green, those of the conjunctiva yellow. At the same time it is often found that the loss of corneal substance is much more extensive than it had appeared under focal illumination. The details can thus be better recognized, and confusion of herpes of the cornea with phlyctenular or eczematous keratitis, with ordinary corneal ulcer, with epithelial erosions, with superficial punctate keratitis, or with vesicular (bullous) keratitis is positively prevented.

Epithelial erosions heal without leaving a trace. Hyperæsthesia or anæsthesia of the cornea is absent. In eczematous superficial keratitis there is considerable reaction of the bulbar conjunctiva. Superficial punctate keratitis occurs often in influenza, with catarrhal irritation in one or both eyes. In herpes or erysipelas there are small gray spots, irregularly arranged in the superficial layers of the cornea, which produce a dull, punctate appearance. Powdering with calomel will cause them to disappear after a few weeks or months. Irritation of the iris, if present, is relieved by instillations of scopolamine. The resulting central maculæ, however, may lead to considerable reduction of visual acuity.

Vesicular (bullous) keratitis is due to degeneration. The corneal epithelium is raised in the shape of vesicles with limpid contents, or of larger fluctuating ones, which soon burst. In most cases the affection is a sequel of absolute glaucoma.

The prognosis is relatively favorable, if the affection has been early recognized and treated. There are no relapses, unless the causative disease recurs.

Treatment.—In my experience, the recovery is most quickly effected as follows: Begin with a fluorescein instillation and touch the vesicles and herpetic ulcers with concentrated tincture of iodine (inspissated to one-half to one-third of the original solution); apply a well-fitting, moist dressing (cataplasma Langlebert, etc., p. 332). This procedure may have to be repeated several times.

A. v. Reuss uses electrolysis with good results. Having stained the ramifications of the herpetic ulcers with methylene blue, he applies along the grooves a platinum electrode, the size of a pin-head.

If increase of intra-ocular pressure is suspected, pilocarpine (equal parts of 2 per cent. Gm., and morphine $\frac{1}{8}$ per cent. Gm.) is instilled. Otherwise pain and subsequent iridocyclitis are relieved by instillations of atropine, as required. A little dionin powder, applied to the conjunctival sac, will also relieve the pain. In the presence of iridocyclitis and hypopyon, heat is applied at regular intervals.

So-called irritative remedies, which serve to increase the fluid, are used to clear up any persisting corneal opacity consisting of cicatricial

connective tissue. They will also aid in preventing a relapse. Owing to the fact that the eye soon becomes accustomed to this irritation, the various remedies are used alternately and continued for several weeks, as may be necessary. Aside from inspergation of calomel, application of yellow salve and iodoform ointment, instillation of potassium iodide (pot. iod. 0.4, sodium bicarbon. 0.2, distilled water 10.0), of equal parts of turpentine oil and olive oil, of dionin 5 to 10 per cent. (Plate II, Fig. 3), tinct. opii crocata, the same remedy evaporated to one-half to one-third (tinct. opii inspissata), are useful for this purpose. Spraying with hot steam through an atomizer, illumination of the cornea with incandescent light, and radiation of the eye with hot air are other remedies. Sub-conjunctival injections of 2, 3, or 4 per cent. saline solution are used in obstinate cases. It is often possible to sooner or later clear up the corneal scar considerably and to restore visual acuity to some extent. The younger the child, the better will be the result.

Staining the spots with the best India ink or creating an optical pupil should be left to the ophthalmologist. In extensive, centrally-located opacities this will not be resorted to until conservative treatment, as described above, gives no more hope of success; that is, when the opacity no longer shows changes and the eye has become completely devoid of irritation. The power of restoration of the youthful cornea, and the continuous effect of conjunctival stimulation, are so great that there may still be spontaneous improvement after the lapse of a long time. No operation is permissible unless justified by careful functional tests.

None but flat and firm corneal scars are suitable for tattooing; in thin or prolapsed scars the procedure may cause maceration, protrusion, and glaucoma. It may cause a recurrence of iridocyclitis, after a serious attack of this disease has been overcome. For the same reason, a corneal scar with impaction of the iris (so-called anterior synechiæ) must not be tattooed.

Adler and others advise electrolysis to clear up old corneal opacities. The cathode of a direct current of 0.2–0.5 Ma. is applied to the cocaineized cornea for ten or fifteen seconds. The anode is applied to the temple or the nape of the neck. According to indications, this treatment is repeated at intervals of from eight to ten days. The electrode consists of a solid silver cylinder of 7 mm. diameter, and is shaped to the corneal curvature. With the exception of the contact surface, it is covered with an insulating caoutchouc cover and the contact is effected by a drop of mercury which readily adheres to the concave surface of the cylinder. If the opacities are small, the cathode is provided with a small silver or platinum head, which is moved to and fro over the opacities.

Reisinger was the first to attempt replacing the opaque cornea by a transparent substance, but thus far only Zirm and Clausen have suc-

ceeded in effecting complete keratoplasty. In partial keratoplasty (A. v. Hippel) there have been successes as well as failures. Löhlein's procedure consists in most careful transplantation of a flap of cornea, adapting it to the neighboring conjunctiva and taking the greatest care to furnish the same nutritive conditions under which the flap had previously existed. The procedure requires great technical skill, but seems to promise better results than the others.

In any case, such surgical interference is permissible only after it has been definitely established that optical iridectomy, with or without tattooing of the corneal scar, offers no hope of improvement of vision.

2. KERATITIS DISCIFORMIS (PLATE VII, FIG. 4)

Keratitis disciformis (Fuchs) has been observed after infection of the cornea by vaccine poison or variola, herpes febrilis, superficial cornea and injuries without demonstrable cause. It consists of a grayish disc-shaped opacity of the middle corneal layer. The opacity is most dense in the centre—that being the place of the primary epithelial lesion—and is demarcated from the healthy tissue by a grayer margin or by several concentric lines.

The affection persists several weeks or months, is not accompanied by pronounced irritation, but often leads to permanent opacity of the affected region. Symptoms of glaucoma have been observed.

The *treatment* consists in an attempt to clear up the infiltration by the application of heat, using an electrothermophore during the day and an antiphlogistic dressing at night.

In one case I succeeded in effecting an almost complete cure by repeatedly painting the centre of the infiltrate with concentrated tincture of iodine. In addition, for purposes of prophylaxis, I prescribed instillations of pilocarpine-morphine in the evening before retiring.

3. ECZEMATOUS ULCERS OF THE CORNEA (PLATE VI, FIG. 4)

This affection is a scrofulotubercular manifestation, often very obstinate and prone to relapses. It sometimes originates in the corneal periphery as an infundibular, marginal ulcer, secondary to a so-called serous eruption, originally situated on the limbus. It appears as a very vascular marginal infiltration, the semilunar apex of which is displaced below Bowman's membrane toward the centre of the cornea (progressive ligamentous or fasciculated keratitis) (Plate VI, Fig. 6). At other times it arises from the confluence of a large number of small phlyctenules in the limbus, producing an annular ulcer which may be complicated by an extensive areolar infiltration toward the corneal centre. This, however, is not to be confused with superficial vascular keratitis which, together with the phlyctenules, disappears without noteworthy injury to the

corneal tissue. The independent eczematous corneal eruption occurs in a different form. There are either superficial phlyctenules, with perhaps a slight loss of substance, or there is a gray-white or yellow, deep infiltration. If the posterior layers are involved, it may lead to a corneal abscess, consequent iridocyclitis, perforation, and the usual sequelæ. But even without this complication, healing proceeds very slowly, leaving a permanent, visible opacity of the cornea and a corresponding reduction of vision.

Treatment.—In vascular superficial keratitis it is sufficient to insufflate calomel, or introduce yellow oxide of mercury ointment in the conjunctival sac. New and superficial eruptions, attended by photophobia, are best prevented by atropine instillations, protecting the eyes from excessive light by smoked spectacles. Fresh, large marginal pustules are energetically painted with concentrated tincture of iodine. The infundibular marginal ulcer requires instillations of physostigmine to prevent perforation (0.05 : 10.0), and a duplex bandage. In impending perforation, with distinct constriction of the pupils due to physostigmine, the ophthalmologist may find it advisable to open carefully the base of the ulcer by galvanocautery.

In order to prevent detachment of the lens by too rapid evacuation of the aqueous humor, and to protect the lens and iris from injury, the galvanic needle should be heated to a light red point, which is allowed to act for a moment, before penetrating deeper. In the presence of hypopyon, a single cauterization of the ulcerous base may be sufficient to effect evacuation of the aqueous humor. Cauterization is interrupted as soon as a serous or purulent droplet appears at the base of the puncture.

Cauterization of the other areas, where there is the loss of substance, is injurious if the ulcers are large, as it may be the means of causing prolapse of the iris and subsequent staphyloma. The aqueous humor, oozing out of the puncture, helps to clean the corneal ulcer. Rest in bed with a duplex bandage usually effects a prompt cure. The bandage, which prevents any movement of the affected eye, contributes rapidly to a favorable result, unless it causes delirium. In that case it is contra-indicated.

Careful puncture often causes a prolapsed iris to retract. This, with the aid of a duplex bandage, may lead to a solid, smooth, corneal scar.

It requires the services of an ophthalmologist to cover the ulcer with a conjunctival flap. The treatment of all but the simplest forms of the disease is best left to the specialist.

In fasciculated infiltration, massage with highly concentrated yellow ointment is indicated, if the apex of the infiltration is neither raised nor yellow, but rather flat or gray, with a band-like opacity. If the apex of

the infiltration is much raised and yellow, and if there are striated opacities toward the centre of the cornea, it points to a spreading of the process. This must be arrested by careful cauterization under local anæsthesia, especially since Axenfeld has invariably found pneumococci at the apex of the infiltrate. General anæsthesia is rarely necessary.

I effect this cauterization by painting with liquefied carbolic acid, using a small, finely-pointed brush, and applying the substance to the affected region until it is distinctly white. The fascicular infiltration is not touched. So long as the eye is not moved and none of the carbolic acid comes in contact with the conjunctiva of the lids, this procedure is quite painless, if carefully executed. This is preferable to painting with the mitigated silver nitrate stick, as practised by Horner, because only those parts are cauterized which are in immediate contact with the brush—provided, of course, that the latter is not overcharged with the carbolic acid. Any excess is removed by first applying the brush to a piece of blotting paper. Carbolic acid penetrates more effectually to the deeper parts of the affected zone, and it is superior to galvanocautery, because the heat radiated by the latter spreads beyond the point of cauterization.

In annular ulcer, the eczema of the lid margins demands attention (comp. p. 89); but as there is usually eczematous or oedematous catarrh of the conjunctiva, the copious secretion is first removed by painting the everted palpebral conjunctiva and fornix carefully with absolutely neutral lead acetate (0.5–1.0 : 30.0). The eye is then carefully cleansed, so as scrupulously to protect the cornea from lead. Or the conjunctiva may be painted with a silver acetate solution, or the stronger silver nitrate solution (0.25–0.5 : 30.0). A duplex bandage, which, if necessary, is moistened at frequent intervals with a 3 per cent. boric acid solution, may be applied over night.

Horner observed grave areolar infiltration in cases where the cornea was almost entirely infiltrated; this condition was removed in two or three days by a firmly applied pressure bandage, “so that there was practically no noteworthy opacity left.”

“The pressure bandage is firmly applied. The palpebral borders being usually eczematous, they are first painted with silver nitrate; the lids are covered with a small piece of linen, over which salicylated cotton is applied. A flannel bandage is then bound over it very tightly. The bandage is renewed twice daily, especially if there are excoriations and eczema of the lids. A wet salicylic bandage may be used, instead of the dry one. The cotton is moistened with a cold, saturated solution (1 : 300), and by pouring some of the solution over the bandage from time to time its antiseptic effect is constantly renewed.”

Independent, superficial, small or large phlyctenules and pustules are found, either isolated or multiple, in the medial parts of the cornea.

The sensitiveness of the injured trigeminus ends is reduced by atropine (0.1 : 15.0), using every precaution to avoid atropine poisoning, to which infants are particularly liable.

Deeper, abscess-like infiltrations may soon be complicated by iridocyclitis and hypopyon. They likewise require atropine. During the day an electrothermophore of chamomile cataplasms is applied (comp. p. 219). At night both eyes are covered with Langlebert's cataplasm or an antiphlogistic dressing.

When the ulcers begin to heal, instillation of fluorescein will indicate the fact. As soon as the irritation of the superficial trigeminus endings, the photophobia, and the spastic contractions of the orbicularis decrease, yellow ointment, freshly and carefully prepared, is applied daily to the lower part of the conjunctival sac. An amount from the size of a hempseed to that of a small pea is used until the eruptions are healed and the vascular repair work has disappeared.

According to Goldzieher, phlyctenular corneal infiltrates which extend to the deep parts should also be looked after. They are accompanied by softness of the globe, and may run an exceedingly dragging course. They usually occupy the centre of the cornea, and gradually fuse with the transparent tissue, leaving indistinct margins. In other cases they are irregularly shaped, confluent gray infiltrations, centrally situated, with evidences of vascular superficial keratitis; in others, again, the phlyctenular infiltrates are accompanied by corneal scars and anterior synechiæ. The anterior chamber is shallow, the pupil considerably contracted, and the iris greatly discolored. As a rule, there are increased lachrymation and ciliary neurosis. Mydriatics, even if repeatedly instilled, will not dilate the pupil. All this suggests that the cornea allows the aqueous humor to escape through a corneal fistula, which relaxes the tension in spite of increased secretion.

Treatment.—Nothing short of repair of the fistula will effect a cure. First, a duplex bandage is applied to keep all pressure away from it, and is continued for eight or ten days. If this is unsuccessful, the fistula is superficially and carefully cauterized with a galvanocautery needle (Plate XXI, Fig. 10), local anæsthesia being applied for the purpose. A duplex bandage, renewed daily, is applied for several days.

As improvement sets in, the tension of the globe increases, and a mydriatic, which had so far been prevented from entering the anterior chamber by the exuding aqueous humor, can now exert its effect. The hyperæmia of the iris and the ciliary neurosis now gradually abate. As soon as they have completely subsided, vascularization has begun and the crusts have fallen off, irritative remedies are applied in the way described.

There are, however, cases of eczematous conjunctivo-keratitis which, owing to unfavorable home conditions, etc., suffer frequent

relapses. This is due to chronic rhinitis and chronic oedematous or pachydermal swelling, usually of the upper lid, which defies even the most careful treatment. Waters containing salt and iodine may effect a cure.

In scrofulous processes of the cornea, subconjunctival injection of air is reported to have been successfully employed, the air being sterilized by aspiration through a tube made incandescent in a spirit flame (Terson). This process requires further testing.

According to Heddæus, scrofulous corneal affections heal much better and more thoroughly with the internal administration of mercury than with any other medication. In mild cases calomel is given in the shape of powder, the dose being a milligramme for each month of the child's age, or a centigramme for each year. The maximum dose is 0.05. In grave cases the inunction treatment is indicated in spite of initial exacerbations. (Also compare pp. 91 and 145.)

4. PANNUS OF THE CORNEA (KERATITIS PANNOSA)

This is a subepithelial, grayish, connective-tissue infiltration of the cornea, which is chiefly permeated by branches of the vessels of the superficial net of marginal loops. It principally occurs in relapsing ulcerative or obstinate corneal processes.

Differential Diagnosis.—The pannus in relapsing eczematous keratitis spreads in sectors, irregularly and in different directions. The typical trachomatous pannus, on the other hand, forms numerous small superficial infiltrates and ulcers, which nearly always spread in a falci-form shape from the upper corneal margin. They are much less frequently found in the lower margin. Should the cornea be mechanically irritated, as in displacement of the cilia, it may penetrate into deep parts (pannus crassus). The conjunctiva, notably the superior tarsal one, undergoes granular cicatrization.

Eczematous and trachomatous pannus also occur together.

Degenerative pannus is the vascular connective-tissue proliferation which occurs at various parts of the corneal border in eyes that have lost their visual power through iridocyclitis or glaucoma. These proliferations may also be due to superficial corneal defects. They may represent a reactive and reparatory process, as, for instance, the epaulette-shaped pannus in parenchymatous keratitis, which is sometimes situated superficially, and sometimes in the deeper parts. They may undergo involution, as in eczematous pannus, to such an extent that no traces can be detected by superficial inspection. On the other hand, intractable trachomatous pannus often leads to keratectasia, with secondary glaucoma and keratosis of the cornea (xerophthalmos).

Successful treatment can be given only in a hospital. Cure of the underlying cause will arrest the vascular new formations. Thus, it will

be necessary in pannus and eczematous-trachomatous keratitis to supplement or alternate the trachoma treatment by local treatment directed against the eczema. Fresh crops are treated with ice compresses to be applied from fifteen to thirty minutes three or four times daily, also cocaine-byrolin salve or novocaine-suprarenin salve (novocaine 0.1; suprarenin. boric. 1.0 : 1000.0-0.5; amyloglycerine 10.0). If the iris is irritated, atropine-morphine ointment should be used. In corneal ulcers the remedies mentioned on p. 196 should be tried, in order to clear up the cornea.

5. MARANTIC NECROSIS OF THE CORNEA (KERATOMALACIA)

This infection occurs in infancy, chiefly as a sequel to typhoid fever, measles, scarlet fever, and variola. It is also found in general tuberculosis, congenital syphilis, diseases of the brain and meninges, in profuse diarrhoea or serious digestive disturbances due to exclusive vegetable diet, and in prolonged sickness. Debilitated children, and those whose nutrition is defective in the first months of life, are usually affected. If the condition continues, the disease may occur at a later period as well. As a rule, these infants are brought up on artificial food.

Ocular affections, even slight ones, favor development of necrosis. It may be preceded by corneal xerosis which has spread from the scleral conjunctiva. Conjunctival xerosis, with corneal disintegration in one eye, may occur without involving the cornea of the other eye.

The corneal affection is bilateral, as a rule, running the same course in both eyes. In the initial stage the lower half of the cornea exhibits a diffuse or dirty yellow opacity. If the epithelial lining is still present, it will be found rough, irregularly stippled, dry and loose. The transversely oval opacity of the lid-fissure zone of the cornea is soon replaced by a loss of substance with a gray-yellow-white discoloration of the base, which extends over the entire depth and surface of the cornea and may early perforate, causing prolapse of the iris. Again, milky-looking shreds of the cornea may become detached, leading, after the lapse of a few weeks, to perforation of the cornea with prolapse of the iris. This happened in the right eye of one of our cases, in which the shreds measured 5 mm. in diameter and about 1/20-1/10 mm. in thickness. Perforation did not occur in the left eye. Later the peripheral parts of the cornea cleared up. An adherent leucoma developed in the right eye, and in the left a central macula. The illness was of about two months' duration.

The scleral conjunctiva varies in color between dark bluish-red, pale yellow, and pale pink; or it resembles the surface of the cornea, is dry, like epidermis, and is covered with a whitish fatty substance, to which tears will not adhere. As soon as complications with iridocyclitis, hypopyon, or panophthalmia set in, as nearly always happens in grave

cases, the bulbar conjunctiva becomes distinctly redder, especially in the lower half of the cornea. Slight œdema of the lids, redness of the palpebral conjunctiva and the fornices will follow, with moderate, mucopurulent secretion, which later changes to a clear, yellow, serous one. The lids are not closed during sleep, but rather gap, so that in deep sleep the lower half or the corneal centre is more or less visible.

Keratitis in lagophthalmos affects the cornea chiefly by the large general loss of water, and is distinguished from the condition just described by the undisturbed closure of the lids. Malacia of the cornea occurs, while the general condition of the patient is still good, but in pedatrophia (tabes mesenterica) it is distinctly dangerous to life, particularly in infancy. Marantic corneal ulcers occurring in measles, scarlet fever, diphtheria, in the exsiccating stage of variola or the later stages of typhoid, run a less rapid course. They are often complicated by abscesses in other parts of the body, which usually begin with a central infiltration and change to ulcers. There is considerable injection of the conjunctiva and ciliary vessels, photophobia, more or less pronounced ciliary neurosis, and often great irritation of the iris, with turbidity of the aqueous humor and hypopyon.

The origin of these manifestations is partly ectogenous. A physician, for instance, accidentally thrust a finger into his eye while performing vaccination, and, although he washed the organ out immediately, a violent inflammation of the cornea developed, leading to extensive opacity. According to Schreiber, insufficient regeneration of the cornea following loss of substance may also be a factor, and be present several weeks before the first symptoms of pedatrophia.

Treatment.—The prognosis of infantile keratomalacia is unfavorable, owing to its precipitate course. In very young children the mortality amounts to about fifty per cent. Nevertheless, the treatment is sometimes moderately successful. Horner succeeded, in a case of hereditary syphilitic keratomalacia, in preserving the cornea with a large leucoma, in spite of perforation. His treatment was a general mercurial one and application of an occluding bandage. An important part of the treatment is improvement of the general condition by attending to the digestion, heart and diet. The internal use of quinine has also been favorably mentioned. Local: Protection of the bulbar surface by a moist duplex bandage in order to guard the cornea against further desiccation; inspergation of a non-irritative, finely pulverized, germ-free antiseptic, like airol or iodoform, by means of a small calcinated spoon once or twice daily; and, as soon as the infiltration has been somewhat arrested, application of heat. In order to stimulate circulation the heat should be uninterrupted, except when necessary.

Moist heat applied by cataplasms, as described on p. 219, is partic-

ularly worthy of attention, since they preserve the heat longest. Nor do they, when continuously applied, cause eczema of the palpebral cutis, like aqueous, boric, and similar compresses.

Lineal cauterization of the fornices with a finely-pointed silver nitrate stick is often advantageous in the milder forms of corneal necrosis, especially if the infiltration and swelling of the palpebral conjunctiva are rather pronounced. I use for this purpose a stick of 90 parts silver nitrate and 10 parts silver chlorate. It can be much more easily smoothed and pointed than the ordinary silver nitrate stick, by washing it with moist cotton. The most practical method is to cauterize first the lower fornix, it being easily accessible by drawing the lower lid downward, while the patient looks upward. The stick is held in an upright posi-

FIG. 30.



Lineal cauterization of the lower fornix.

tion, and the point is drawn gently and rapidly along the entire length of the lid (Fig. 30). A previous instillation of alypin will serve to prevent movements of the eye. The scab need not be neutralized with a saline solution if the lid is kept away from the eye for a few seconds after cauterization. Owing to increased congestion, the cautery at first causes an apparent exacerbation, and it is not to be repeated until the scab has completely desquamated and the epithelial lining of the conjunctiva has undergone restoration and the cauterized places again look as before.

6. KERATITIS FOLLOWING LAGOPHTHALMOS

This affection has been observed in paralysis of the facial nerve after influenza and measles, in epidemic cerebrospinal meningitis, and in other acute infectious diseases which are characterized by stupor. The corneal reflex is diminished, and the reflex lid movement is absent.

The evaporation causes dryness and later ulceration of the cornea. As patients are prone to keep their eyes turned upward, the uncovered part of the bulbar conjunctiva becomes a dark red and is permeated by ecchymoses. Any conjunctival secretion dries into crusts at the palpebral borders. In the lower part of the cornea some dry gray or brownish-gray scabs will be formed and desquamated in a few days, leaving an ulcer which extends to the deep as well as the superficial parts. In favorable cases this will heal with corneal leucoma, but may also lead to hypopyon, iridocyclitis, and phthisis bulbi, provided there is recovery.

The *local treatment* in grave cases consists in closure of the lids with adhesive plaster, the strips being fastened at right angles over the lids, which have been firmly approximated. A moist bandage is then applied. In milder cases a bandage is applied over night, and unsmoked protective spectacles are worn during the day. Paralysis of the facial nerve is treated with the electric current, about which compare chapter on "Affections of the External Ocular Muscles." In incurable paralysis of the facial nerve, shortening of the lid fissure by tarsorrhaphy may have to be resorted to.

7. KERATITIS NEUROPARALYTICA

This condition is the result of paralysis of the trigeminus or fifth nerve. It is a sequel of faucial diphtheria, herpes zoster, diseases of and operations on the gasserian ganglion, and marantic and septic thrombosis of the sinus cavernosus. Reflex secretion of the lachrymal fluid is destroyed, so that the affected eye remains dry when the patient weeps. The cornea, deprived of its normal secretion, becomes dry. The initial opacity of the anæsthetic cornea is soon followed by desquamation of the epithelium, which progresses from the centre toward the periphery. The centre of the cornea may break down and slough. Aside from ciliary injection, there is but slight evidence of inflammatory irritation.

The *prognosis* is doubtful, because, in spite of the most careful treatment, there remain an extensive, dense corneal scar, flattening of the cornea and perhaps impaction of the iris and staphyloma, with considerable impairment of vision. In one of my cases, following removal of the gasserian ganglion, there was a cure with perfectly normal vision by the mere application of heat and a watch-glass bandage. In secondary iritis, instillation of scopolamine-atropine is required. Electric treatment and subcutaneous injection in the temporal region of strychnine (0.001–0.003) have been favorably mentioned. Sufficiently large protective spectacles should be worn continually during the day, in order to prevent relapses. For the same reason very careful after-treatment is required.

8. SEPTIC SUPPURATIVE KERATITIS (SERPIGINOUS ULCER OF CORNEA)

There are three forms of this affection: (1) Ulcer, which is usually stationary. In this type there is a superficial, disc-shaped defect, with yellowish-gray infiltration, which clears slowly. Its cause is attributed to infection with diplococci, or, when the ulcer is located at the margin, to the bacillus of Nedden. (2) A superficial defect with a number of isolated, subepithelial infiltrates, the so-called streptococcus ulcer. (3) A progressive suppurative ulcer, which has nearly always been preceded by an injury to the corneal epithelium from an infected foreign body, or from a contusion. (*Ulcus corneæ serpens*, *hypopyon keratitis*, *pneumococcus ulcer*; Plate VII, Fig. 3.) Serpiginous ulcer in *varicellæ*, *variola*, *measles*, etc., is likely due to a corneal abrasion with a finger-nail. Old affections of the lachrymal sac and conjunctiva, which are a frequent causative factor in adults, practically do not exist in children.

The typical angular form occurs only exceptionally—a point of importance in making a diagnosis. Contrary to the pathological picture in adults, serpiginous ulcer in children occurs as a puffy infiltrate, which protrudes above the corneal surface. Its color alternates between uniform white and yellow. The color may be more intense towards the centre, in which particular it differs from the picture in adults, where the centre of the ulcer is less opaque. E. Fuchs attributes these differences to anatomical conditions: "The cornea of young persons has a great tendency to puff up in inflammatory conditions. This explains the considerable size of the infiltrated area of the cornea. The increased opaqueness in the centre is clearly due to the fact that the pus-cells have penetrated to that point, while in adults only a small number of them invade the necrotic focus. It is probable that the leucocytes emigrate from the marginal vessels of the cornea more rapidly in children than in adults. The distance the irritative substance must travel from the focus to the corneal border is less in children, and this is true also of the distance the leucocytes must travel from the corneal border to the infected focus. Possibly also the motility of leucocytes in children is greater than in adults."

The suppurative infiltrate, which is at first annular and rather sharply demarcated, soon assumes the shape of a disc, and has a tendency not only to penetrate into the corneal parenchyma, but also to spread along the surface. It is at first a yellowish, falciform infiltration of only one marginal part and in one definite direction.

The deep, scrofulous infiltrate is not sharply demarcated, is sometimes a pus-colored yellow, often does not disintegrate at all, and therefore leaves only an insignificant opacity. Serpiginous ulcer is differentiated from the latter by the fact that, even with a slight loss of corneal

substance, toxic iridocyclitis develops at an early stage and is sometimes accompanied by violent ciliary pain, cloudiness of the aqueous and hypopyon. The hypopyon of serpiginous ulcer may occupy, according to circumstances, three-fourths or more of the anterior chamber. A rapid increase in the size of the ulcer, both in the deep and superficial parts of the cornea, will decide the question.

At the very onset of serpiginous ulcer there is often inflammatory oedema of the bulbar conjunctiva, soon followed by oedema of the lids. As the corneal suppuration progresses and develops into panophthalmia, there is inflammatory infiltration of the peribulbar tissue, with protrusion of the globe. The ulcer threatens principally the centre of the cornea, and thereby the visual power, to a considerable extent.

In my opinion it is a matter of importance whether the cornea has suffered a serious contusion by foreign bodies, etc. If so, there is instant interruption of the corneal nutrition at the injured point, and with it detachment of the loosened epithelium, which cannot but favor infection. The more or less continuous contact of the injured corneal region with the virulent contents of the conjunctival sac is of equal importance. Indeed, the disc-shaped opacity in the very first stage of serpiginous ulcer depends materially upon an unevenness of the epithelium. The connecting link between infection and the primary injury is a vesicular, circular epithelial detachment. After this has ruptured, the subepithelial infectious matter causes the formation of an arcuate infiltration in the border of the epithelium, which it undermines. For this reason, then, early removal of the epithelium, and painting of the ulcerous base and the marginal infiltrate with tincture of iodine, will soon arrest the pathological process (Edmund Jensen).

Differential Diagnosis.—As compared with simple corneal ulcer, the area surrounding serpiginous ulcer shows more or less breath-like striæ. At an early stage there is secondary inflammation in the iris and ciliary body.

The *prognosis* of the first two types is relatively favorable, while in progressive purulent ulcer it is perhaps somewhat less favorable in children than in adults, owing to the anatomical conditions of the cornea already described. The outlook is doubtful, even in cases which promise well in the beginning. But the earlier the ulcer has been recognized, and the earlier treatment is instituted, the more hopeful is the prognosis.

Treatment.—Stationary ulcers, which are often accompanied by hypopyon, are touched with ordinary or concentrated tincture of iodine. This procedure may be necessary several successive days. In addition, instillations of atropine and application of a binocular bandage are used (Fig. 31). Repeated irrigation of the cornea with $\frac{1}{2}$ per cent. solution of zinc sulphate or mercury oxycyanate (1.0 : 5000.0) has been recom-

mended; also iontophoresis with $\frac{1}{2}$ per cent. zinc sulphate which is applied once or several times, according to conditions, by a current of 1 Ma. for one minute or of $1\frac{1}{2}$ -2 Ma. for two minutes, the smallest electrode being used for the purpose (No. IV). According to Wirtz, it is of the greatest importance to use perfectly pure distilled water in preparing the solutions.

In progressive purulent ulcer, previous to the search for pneumococci, the lachrymal secretion is examined by exact rhinological tests, if necessary. Repeated palpation of the lachrymal sac is indispensable, since in an abrasion of the posterior wall of the sac the secretion exudes posteriorly instead of toward the puncta. It may even require the application of a protective bandage over night to arrive at a positive result.

It then depends upon the kind and quantity of the secretion, on the consistency of the conjunctiva—which may exhibit chronic catarrh—and the appearance of the corneal ulcer, whether immediate external incision and tamponade with iodoform gauze, or extirpation of the lachrymal sac should be resorted to, or whether systematic expression and irrigation of the sac with an antiseptic solution is permissible. If the ulcer is still in an early stage, and shows no tendency to spread, conservative treatment may be tried. This consists of rest in bed, instillation of atropine twice daily, irrigation of the ulcer with undiluted and freshly prepared chlorine water, and in the inspergation of iodoform, dermatol, airol, xeroform, euophen, either alone or together with equal parts of pulverized boric acid, etc. Compresses moistened with 2-5 per cent. hydrogen peroxide give great relief. They are first applied cold and gradually warmer, and are changed hourly according to indications. A similar bandage is applied during the night. It has an inner layer of gauze covered with gutta percha and provided with an opening, through which a fresh supply of hydrogen peroxide is administered.

Some practitioners, including Arens and Heilbern, have found copi-

FIG. 31.



Binocular bandage.

ous instillations of pyocyanase very efficacious. "It has a dissolving and destructive effect not only on cultures of *Bacillus pyocyaneus*, but also on the pneumococcus and other heterogeneous microorganisms." It is applied every half hour. In order to retain it as long as possible, the eyes are kept closed.

I also paint the infiltrated marginal area with concentrated tincture of iodine several times a day for several days in succession, until there is a distinct arrest of the ulceration, and an intensely brown coloration appears. This procedure is both simple and painless and, if energetically applied, will prove as effective as painting with carbolic acid, sub-conjunctival saline injections or iontophoresis, or galvanocautery. It has an advantage over the cautery, as the latter does not expose the non-affected tissue to the effects of radiated heat. The method may be continued, if after twenty-four hours the infiltrated border has not spread, and if after repetition it begins to disintegrate. Isolated fresh infiltrates may appear at the margin of the painted zone for one or two days later, but they are rapidly destroyed with renewed iodine painting. The persisting corneal opacity is more delicate than that attained by galvanocautery.

Should the ulcer, nevertheless, progress, transverse incision after the method of Saemisch is done. This applies to larger ulcers also, in which painting is ineffective. A second incision, parallel or vertical to the first, is rarely necessary. Saemisch demands aëration of the wound twenty-four hours after operation, and attaches great importance to it, because he often observed that it was conducive to a definite arrest of the ulcerous process. I rather prefer to do without it, in the interests of smooth healing and to prevent adherent leucoma. At the same time, I precede the transverse incision by a temporary blepharotomy, by liberally incising the external canthus for 1-2 cm. with blunt scissors. Hemorrhage of the severed palpebral vessels is spontaneously arrested in a short time, but as it accelerates the elimination of the toxic substances from the iris and ciliary body, I do not interrupt it. For the same reason I keep the wound open for several days, by separating the edges. It is only in exceptional cases that the bleeding vessels have to be ligated or sutured. Afterward, if at all possible, a binocular bandage with Langlebert's cataplast, is applied and changed every twenty-four hours.

In very advanced stages of the ulcer, neither cauterization, iontophoresis, nor transverse incision is advisable. The latter is objectionable, because the great extent of the incision may lead to prolapse of the iris. The operation should rather be limited to careful puncture of the anterior chamber at the attenuated point of the ulcer by means of galvanocautery. This has often enabled me at least to preserve part of the cornea.

Should the other eye be already destroyed (by phthisis bulbi, etc.),

and the opacity occupy the entire centre and lateral parts of the cornea, an optical pupil may have to be considered, but only for the upper segment of the iris. In such cases I precede iridectomy by a backward displacement of the superior rectus muscle, in order to render the incidence of the light more favorable.

9. PARENCHYMATOUS KERATITIS

(SYN: KERATITIS INTERSTITIALIS PROFUNDA, DIFFUSE CORNEAL INFILTRATE, PLATE VII, FIGS. 5-7)

This disease is due to an endogenous infection, and is usually a sign of syphilis, either inherited or acquired at birth, or of tuberculosis.

Syphilitic keratitis rarely occurs before the sixth or seventh or after the twenty-fifth year. In most cases it sets in before puberty. The girls are more frequently affected, than boys. In many cases the early evidences of syphilis are present in the first years of life, but are so trivial as to escape notice. The corneal infiltrate may also develop in eyes which had been infected with iridocyclitis during uterine life, but it is very rarely observed at birth or in the following few months. Both eyes are often involved simultaneously or in rapid succession: a point to which the attention of parents must be called. In acquired syphilis an infiltrate does not develop very often, but if it does, it is usually confined to one eye.

The clinical picture varies. The corneal infiltrate may start in the centre or at the margins, but it nearly always invades the entire cornea. This extension may be gradual or sudden. It consists of cloudy, rod-shaped, whitish-gray, light gray or delicately transparent grayish or yellowish-gray opacities, which are not uniformly dense and are situated in various parts of the cornea. The infiltration is usually most dense in the centre of the cornea. The opacity may also be confined to certain quadrants or marginal zones. It may also be so pronounced as completely to cover the cornea, decreasing the visual power, or impairing it to such an extent that hand movements can be recognized only. Conjunctival and pericorneal injections are not very pronounced. The subjective manifestations, such as photophobia, lachrymation and pain, are slight. They increase, however, when the iris and ciliary body are much inflamed, leading to blepharospasm, severe ciliary pain, irritation, and slight elevation of temperature. The corneal surface is dull and delicately spotted, giving the appearance of a dimmed pane of glass. When the opacity is considerable, the corneal surface resembles a dull, opalescent glass.

The infiltration is rarely enough to cause vesicles and threads, lifting the corneal epithelium. Involution of the infiltrate usually starts at the corneal margin, vessels budding in from the marginal net loops through all the layers of the cornea from above downward, arranged in

a brush-like or net shape (Plate VII, Fig. 5). Their remnants, in the shape of delicate, whiskbroom-like ramifications, can often be seen in the corneal tissue with the naked eye or a loupe, even after the lapse of several years (Plate VII, Fig. 7). These resorbing vessels either cover the entire cornea or leave the centre free. They lie so closely together that they look like a salmon-colored, red-gray or cherry-red, hemorrhagic surface, arranged in the shape of epaulettes, protruding in staircase fashion above the level of the anæmic corneal parts (pannus). In other cases they are closely squeezed together and, pushing the opacity before them, come to an abrupt halt in a sharp line near the centre of the cornea. The opacity gradually clears up from the margin toward the centre. The clearing process may be rapid or it may occupy months or years. Gradual elimination of the opacity without vascularization has also been observed (Plate VII, Fig. 6). An intra-uterine infiltrate of the cornea causes a deeper, diffuse, dull gray or bluish opacity of the cornea and clears up only to a slight extent or, perhaps, a little better if there is new formation of vessels. This, however, can be detected only with the aid of a loupe. Relapses are rare.

Delicate, gauze-like opacities nearly always persist, notably in the centre of the cornea. The visual acuity is accordingly always more or less reduced, especially when the clarification of the centre of the cornea is arrested, or when the infiltrate undergoes sclerosis, with considerable tissue concentration, and with formation of whitish spots or caseous hyaline flakes and flattening of the cornea. The cornea of one eye may become almost completely clear, while the other eye, owing to contraction and flattening of the cornea, becomes almost totally blind.

In nearly all cases there is hyperæmia of the iris, iritis, or iridocyclitis, which may, however, not be recognizable until the corneal opacity has cleared up. If there are swelling of the cornea and iris, exudative deposits or precipitates, central or peripheral agglutinations between iris and cornea, then anterior synechiæ may develop, even without perforation of the cornea. There may also be a deepening of the anterior chamber. Hypopyon is rare.

Dense parenchymatous infiltration of the cornea also occurs in acquired syphilis, as a sequel of recurrent iritis; there may also be parenchymatous opacity of the corneal periphery in iritis or slight swelling of the iris, but this is rare. Unlike the corneal infiltrate, described above, these opacities, occurring in the later stages of syphilis, remain marginal. General treatment accelerates the cure without vascularization.

Areolar and disseminated chorioiditis, as well as equatorial and peripheral chorioretinitis (Plate XVII, Fig. 1), are of frequent occurrence, and are sometimes so pronounced that the corneal affection appears as a continuation or sequel of the uveal affection, unless it is

directly caused by spirochæta. Vitreous opacity must be suspected in cases where tension is reduced. Even transient and considerable shrinking of the globe, with obliteration of the anterior chamber and temporary blindness, has been observed in these conditions. Atrophy of the optic nerve, with permanent impairment of vision, may also be a sequel of chorioretinitis or neuritis of the optic nerve. It is necessary to examine repeatedly the visual acuity and projection of light. The latter is undisturbed when the cornea alone is involved.

Maceration of the central parts of the infiltrate may cause permanent impairment of vision, by staphylomatous protrusions of the cornea, and lead to total blindness by subsequent glaucoma. In exceptional cases the infiltrate develops into actual suppuration or abscess formation. Yellowish, rod-shaped or striated areas leave persistent delicate scars, or are changed into cystoid, minute, hollow spaces which cause less visual impairment. The keratitis described as *keratitis parenchymatosa punctata profunda* consists of circumscribed grayish areas, the size of a pin-head, in the layers of the corneal substantia propria. These opacities often can not be recognized except with the aid of a loupe, but mere sensitiveness of the eye and slight injection of the corneal net of marginal loops point to the presence of the affection. These infiltrations may develop and disappear rapidly, leaving a punctate opacity behind.

In punctate keratitis complicated with iritis, occurring in the late forms of syphilis, the cornea shows a grayish opacity only in parts. There are circular, light gray or yellowish-gray spots the size of a hemp-seed, located principally in the deeper layers of the corneal parenchyma, in some places isolated and confluent in others. I have also seen this form as an early symptom of the disease.

Annular keratitis is not a very frequent variety of hereditary congenital parenchymatous keratitis, and occurs almost exclusively as a transient stage of the latter. The centre of the cornea is more transparent, and surrounded by a very opaque annular zone consisting of several confluent infiltrations. If the lower parts of the cornea are chiefly attacked, it is not always easy to decide whether there is merely a corneal affection, or an iridocyclitic exudate of the anterior chamber, with an opacity of the deeper corneal layers.

All these infiltrations, as described, are the expression of one and the same pathological process, which varies even in its local manifestations, according to the individuality of the patient and the character of the infection. The fact that the infiltrations are sometimes diffuse and cloudy, and at other times punctate, is probably explained by the deviations in the anatomico-histological structure of the cornea.

These conditions must not be confused with post iridocyclitic

keratitis profunda, which in its onset either resembles that of sclerotic keratitis, or is concentrated in the lowest layers of the corneal centre.

The clinical examination includes not only inspection of the globe with the unaided eye, but also tests of the ocular tension, reaction of the pupils, and the use of the dark room for focal illumination and illumination of the eyeball with reflected light. According to Gallestrand, examination with the perforated plane mirror and Nernst's division lamp is far superior to the ordinary examination with radiant light. The following preparations are used for the diagnostic dilatation of the pupils: Homatropin. hydrobrom. (1-2 per cent.), or mydrin, which consists of ephedrin. mur. 1.0, homatropin. hydrobrom. 0.01, 3 per cent. solution of boric acid 10.0 (Gebbert). In examining outdoor patients, however, a subsequent test for constriction of the pupils by instilling a $\frac{1}{2}$ per cent. solution of physostigmine should never be omitted.

Differential Diagnosis.—Exact history, Pirquet's cutaneous reaction or the diagnostic injection of old tuberculin, the Wassermann reaction, and careful examination of the general condition, are points of the greatest importance. Although typical parenchymatous keratitis is in most cases a sign of syphilis, the clinical picture occurs in a number of other diseases. Thus, Krause observed the rare effect of chrysarobin in the form of a parenchymatous, disc-shaped opacity of both corneæ in a boy who had been treated for four weeks for psoriasis. The clinical picture also occurs in hemorrhagic scarlatinal diphtheria, as metastatic gonorrhœal manifestations, and in grave forms of influenza and malaria. Furthermore, it is seen in childhood as a tuberculous affection with various glandular swellings, and as a sequel to tuberculous infection of the uvea. The latter has also been observed in syphilitic infections. A mixed infection may make the usual treatment for congenital syphilitic corneal infiltration unsuccessful. The efficacy of antisiphilitic remedies, as such, does not prove the absence of tuberculosis, since the latter may also be favorably influenced by them. A tuberculous infection, however, probably does exist, if the subcutaneous injections of tuberculin give a positive reaction, and there are no certain indications for another etiology of the ocular affection. A positive tuberculin test is a certain proof of the tuberculous nature of the ocular affection if there are focal symptoms of the eye, such as ciliary injection, precipitates or nodules in the iris, and fresh hyperæmia of old sclerotic regions. A negative test points the opposite way. The test injections are indispensable for the diagnosis of difficult cases of chronic uveal inflammations. They are best made in the presence of an ophthalmologist, and begin with 0.001 c.c. of old tuberculin, the dose being very gradually increased to 0.0025 c.c. for the seventh injection, when the treatment is stopped (F. Schöler). A. v. Hippel uses 0.004 as second and 0.005 c.c. as third and final injection.

Tuberculosis of the cornea is usually a corneal infiltration with yellowish-white or gray-yellow nodules which, unlike syphilitic parenchymatous keratitis, is initially confined to parts of the cornea. It is nearly always the sequel of tuberculous iridocyclitis. Gray-yellow foci of the limbus in the deeper corneal lamellæ, spreading slowly a short distance toward the cornea, are very indicative of tuberculosis. Direct spreading of the tuberculous focus of the sclera or anterior part of the uvea to the cornea usually causes perforation of the focus outward, with disintegration of the cornea.

Typical acute bilateral infiltration of the cornea is to a certain degree indicative of syphilis. This is an important etiological factor if the Wassermann test is positive, and the history and general physical examination furnish other confirmatory evidence. Repeated abortions of the mother, great mortality of her children, and specific manifestations in other children are valuable indications.

Partial or total deafness, due to affections of the labyrinth and tympanic membrane, anomalies of the intellect (arrest or precocity), recurrent affections of the joints, notably of the knee or foot, osseous defects, periosteal swelling, osteochondritic deformation of the epiphysis of the long tubular bones, elbow, hand, knee or foot joints, causing almost incessant whining in infants, can be verified by radiography. Paronychia of several fingers; protrusion of the frontal eminences; striking flatness of the upper maxillary bones; low or collapsed nasal bridge due to necrosis of the vomer; chronic coryza, ozæna and dacryocystic blennorrhœa, due to deeply-seated affections of the nose; rhagades or lenticular, white, radiating scars in the region of the corners of the mouth, or around the mouth and extending radially to the cheek or frontal region; scars and ulcers, notably of the hard and soft palate, velum, fauces and larynx; dwarfish stature, due to structural osseous affections, etc., are symptoms found in these patients. Moderate swelling and induration of the lymph-glands, especially the cervical ones, and anal papules, are not rare. Other valuable signs are manifestations of congenital syphilis at birth or during the first month, such as coryza, splenomegaly, red discoloration of the skin, papulous, bullous or pustulous syphilides. If the corneal affection develops in the first month of life, there will be spotted, red-brown eruptions of the palmar surfaces of hands and feet, mucosal papules of the gluteal folds, genitocrural folds and anus, and a senile expression of the face.

R. Virchow and G. Lewin emphasized smooth atrophy of the lingual base, due to condensation of the follicular glands, as a symptom of hereditary syphilis.

The third sign in Hutchinson's triad is defective dentition (Fig. 32). The incisors, especially the middle upper ones, are of cuneate form, of

dirty yellow gray color, and, owing to the loss of enamel, eroded in semi-lunar form. There are longitudinal grooves in the lower, and often transverse grooves in the upper part. The interstices are wide, the teeth converge toward the median line of the face, are very small or entirely absent. Hypoplasia of enamel, the defective development of the crowns, and the delayed dentition which often occurs in hereditary syphilis, are no doubt due to rachitis. However, they support the diagnosis of congenital syphilis, if other signs indicate it. Fasini demonstrated spirochæta in the tooth-pulp.

As to the eye itself, Fuchs noticed a vertical oval shape of the cornea

FIG. 32.



Hutchinson's teeth.

in a few cases of keratitis, which he had also seen in congenital syphilis, without signs of present or previous parenchymatous keratitis.

As to the prognosis, fresh outbreaks and relapses must be expected in patients who were not treated early or long enough. Aside from these considerations, the prognosis is the more favorable, the younger the child, the better his general condition, and the less the uvea, retina, and optic nerve are involved. In any case treatment will probably have to be continued for a number of months, and the affection may also occur in the other eye sooner or later. Vision will at first be progressively impaired and for a time there may be almost complete blindness. This refers particularly to cases with extensive vascularization.

For parenchymatous keratitis due to traumatism, see "Injuries."

Treatment.—All cases in which structures other than the cornea

are involved and which, owing to unfavorable hygienic or dietary conditions, cannot be well attended to at home, must be referred to a hospital.

Parenchymatous keratitis due to hereditary syphilis is first treated with mercury and then with iodine. The initial doses should be large, and gradually reduced. Mercury and iodides have a favorable effect in clearing up the cornea, especially in the lighter cases. While the iodides (potassium iodide, syrup ferrous iodide, etc.) are usually well borne, even in hereditary syphilis, caution is required in using mercury, especially if the patients are weakly and anæmic children of lowered resistance.

Rocchini, Balzer, and Fage have successfully injected "enésol" (salicylated arsenious mercury) into the gluteal region, but further tests are required to establish its value.

When salvarsan is used in parenchymatous keratitis of congenital syphilis, there should be a ciliary injection strong enough to maintain its effect for one or two days, while the vessels in the cornea should be moderately congested. It is more than doubtful, however, whether this remedy will positively influence the corneal process, although Löhlein demonstrated experimentally that arsenic was present after twenty hours, even in non-vascularized corneæ. It certainly does not prevent the second eye from becoming affected, as was shown by two cases in our clinic. Furthermore, injections of salvarsan have been followed by grave parenchymatous keratitis in the second and previously normal eye. This occurred also in a non-irritative eye with delicate old corneal opacities, the affection remaining refractory to repeated doses of salvarsan.

One case of bilateral parenchymatous keratitis and iritis showed an exacerbation after ten weeks.

Löhlein found subconjunctival injection of salvarsan without effect, although it had been preceded by gluteal injections.

The most important point is improvement of the general condition and regulation of digestion. Fresh air, preferably at medium altitude, mild diaphoretic (p. 235), and massage of the entire body, are the indicated treatment.

The simplest way of inciting cardiac function consists in the administration of dialysée sudorifique Golaz, 10–15–20 drops to a cup of tea (lime blossom, lilac blossom, chamomile, peppermint, etc.), to which 10–20 Gm. of liquor ammonii acetates have been added. Next comes sodium salicyl., aspirin or novaspirin, 0.5–1 Gm. administered in hot weak tea, with careful packing in woollen blankets and application of hot water bottles, if necessary. Pilocarpine, the most efficacious sudorific, is not without undesirable by-effects, although it has done well in inciting salivation in small doses in so-called tuberculous serous iridocyclitis (p. 247). Hot baths with subsequent dry packs, steam packs with sud-

rific bed apparatus and electric light baths, should be ordered with care. They should be avoided where there is a tendency to cerebral congestion, cardiac insufficiency, atheromatous degeneration of the vessels and chronic renal affections; consequently in intra-ocular hemorrhages dependent upon these conditions.

The gentlest and simplest laxative and diaphoretic measures consist in partial (foot and arm) baths at gradually increasing temperatures. When continued for fifteen minutes, with gradual additions of hot water up to 35–40° C. (90–104° F.), the circulation is regulated and blood-pressure lowered. A roborant tonic dietary should be prescribed, consisting principally of eggs, milk, veal, fowl, soups prepared therefrom, easily digestible fruit and vegetables containing nutritive salts (carrots, spinach, etc.).

The following curative springs enjoy an excellent reputation in the German-speaking countries: The iodine springs at Hall (Upper Austria), Salzbrunn (near Kempten in the Algäu), Heilbrunn, near Biehl, and Tölz (Upper Bavaria); the arsenious springs at Dürkheim, Levico, Roncigno, Guben. The following medicaments are to be recommended: Cod-liver oil, iodide of iron and iodferratose. Iodine, however, is suitable only in the stage of involution.

Local Treatment.—From the onset of the affection, instillation of atropine or scopolamine-suprarenin (1 : 1000, 5 drops to 10 Gm. of atropine or scopolamine solution) should be used, especially if iridocyclitis is impending or present. Atropine must be prescribed free from hyoscyamine, or optically inactive, as demanded by the English pharmacopœia, since atropine intoxication has occurred in children. When the pupil is dilated and the maximum infiltration has been reached, it is usually easy to keep it dilated by less frequent instillations. Atropine should not be used profusely even if dense corneal infiltration allows only traces of it to reach the iris, and the pupil remains contracted in spite of frequent instillations. If there is suspicion of glaucoma, the instillations should consist of pilocarpine-morphine (2 per cent. or 1/20 per cent. $\bar{a}\bar{a}$, according to conditions).

As children have a tendency to keep their eyes closed, to hide themselves in dark corners or to bury their heads in pillows, bandages should be applied sparingly, so as to keep the eyes accustomed to light. It is sufficient to protect the eyes with large, concave, smoked spectacles. Ciliary pain and photophobia are also relieved by applying small quantities of dionin (ethylmorphine chloride) to the inferior tarsal conjunctiva once or twice a day.

Sylla prefers hydriodic ethylmorphine, a white powder which is not easily soluble in water, and should be kept in a dark container. According to Greven, it does not cause such violent burning as dionin. Besides, it seems to have a strong resorptive and antiseptic effect, due to the iodine it contains.

Boric acid applications at room temperature, to be applied three or four times daily for fifteen to thirty minutes with the patient in the recumbent position, afford a pleasant sensation, as do these hot applications:

Hot water compresses are made rather thick, and are immersed in a 3 per cent. borax or boric or borosalicylic solution (salicylic acid 5.0, boric acid 15.0, distilled water 500.0; Sattler). They are applied to the closed lids three or four times daily for fifteen or thirty minutes, or longer. The solution is heated on a spirit regulator up to 35–40° C. (95–104° F.) or, if indicated, to 45–50° C. (113–122° F.). The compresses are squeezed out, so that they no longer drip, and changed at regular intervals.

Warm Chamomile Compresses.—Small compresses immersed in the infusion must be absolutely clean. They are covered with flannel and changed every few minutes. Hot chamomile infusions also have a certain antiseptic value, owing to the terpenes they contain. Linseed cataplasms have a higher temperature and therefore retain moist heat longest, so that they need not be changed so often as hot water compresses. Nor will they cause cutaneous eczema, which is quite unavoidable in continuous application of moist compresses. Correctly prepared, cataplasms are just as aseptic as the others, because, having been boiled down to a thick pap, infectious germs are destroyed. Clean and carefully sterilized linen is used as a cover.

Dry heat is applied by means of Japanese warming boxes (p. 74, Fig. 17). They are filled with a specially prepared, pulverized, compressed coal which will glow for about two hours. The box is covered with moist cotton where it comes in contact with the eye, and fastened in the proper position with a gauze bandage in the shape of a monocus.

The so-called thermophores consist of a rubber bag, closed with a screw cap. They contain thiosulphuric sodium, dissolved in hot water, which liberates heat upon recrystallization.

The electric thermophores (Salaghi, p. 74, Fig. 16) are of lighter weight and applied to the eye on a dry layer. The desired degree of heat is exactly controlled by rheostats or by a series of resistance lamps.

The moist warm applications are applied as follows: The lids and adjacent parts are cleansed with sterilized cotton or gauze pads. A piece of cloth, about four inches in diameter, is saturated with 3 per cent. boric acid or 1 per cent. acetate of aluminum, and applied to the eye. It is then covered with a piece of gutta-percha paper or Billroth's batiste fastened to the forehead and chin by means of small strips of adhesive plaster or leucoplast. Over this is placed a bandage, of gauze or etamin. On removing the bandage the place of application is cleaned with boric acid, to protect it from maceration.

An excellent moist, aseptic compress is the cataplasm of Langlebert,

which is easily and conveniently applied, soft and flexible, and retains the moist heat without irritating the skin. It may even be allowed to remain for the whole day. I have had a chamomile cataplasm prepared on the same principle, which is very suitable for a "permanent dressing."

Antiphlogistine serves to induce active hyperæmia. It is a paste, which, according to the manufacturers, consists of an American silicate alumina and chemically pure glycerine, with the addition of small quantities of antiseptic substances (boric and salicylic acid) and traces of iodine and volatile oils. It is non-irritating and absorbs water freely, and, so far as the eye is concerned, I have found that it retains heat and moisture uniformly for twelve or fourteen hours. The paste is applied with a spatula, and covered with a thin layer of absorbent cotton or gauze. As soon as it has lost its efficiency, it drops off of its own accord.

Giese and Hertel have established that warm compresses raise temperature in the conjunctival sac by more than one degree. Heat, therefore, indirectly incites the circulation of the blood in the anterior globe. By supplying fresh and healthy nutritive and building material, it helps to eliminate the toxic substances in the cornea, sclera, iris and ciliary body, thereby promoting the desquamation of dead corneal tissue and the resorption of corneal infiltrations. In iridocyclitis and iridocycloscleritis the venous stasis in the plethoric anterior uveal section is diminished by conducting the blood to the periphery. This is shown also by the dilatation of the pupil, which occurs after prolonged application of heat to the eye.

As a rule, the application of absorbent dressings, warming boxes, and cataplasms is not borne well until the beginning of vascularization. As soon as this is fully established, the process can be considerably shortened by daily application of a few granules of dionin. In cases which run an atonic course, resorption should be incited by alternate superficial touching of the upper and lower fornices of the conjunctiva with a uniformly smooth and very finely pointed stick of silver nitrochlorate as described on p. 205.

So long as corneal ectasia is impending or present, it is necessary to apply a duplex bandage (p. 209, Fig. 31). It is applied as follows:

The child is put to bed, gently closing his eyes as if asleep. An oval piece of sterilized gauze is applied over the lids, completely covering them. The area between the superior orbital border, cheek, and eye is filled out with pieces of absorbent gauze or cotton, so as to form a cushion of one or two inches, extending beyond the lids in all directions. This is covered by a bandage of gauze or elastic, large-meshed, cotton, two inches wide and six or eight yards long, which has been moistened by pouring water over it. It should not be immersed in water, as this would interfere with the unrolling of the bandage. It is then applied to the

more affected eye first, commencing at the lobe of the ear on the same side and proceeding over the middle of the cotton pad obliquely to the opposite side of the forehead. There follows a circular winding closely above the ears, around the occiput and forehead. Continue over the vertex and occiput and under the lobe of the same side, without interfering with its position, and back to the eye. The second oblique winding is applied above the first. A third oblique winding proceeds in the same way below the first; a fourth winding, again following the course of the first, is conducted toward the temples and finally twice around the head. With each winding the bandage is gradually and uniformly tightened. In order to prevent any movement of the affected eye, and also to protect the healthy eye from bright light, the last transverse winding is continued by a few oblique turns from above downward over the forehead, covering the second eye. This, however, can also be done by a separate bandage from below. The position of the bandage may be further secured by reversing the oblique windings above the forehead, when commencing the course over the head. Finally, the bandage is fastened with safety pins at various parts of the circular windings, which are particularly liable to slip upward at the back.

The bandage is changed daily or at longer intervals, as required.

Dense corneal maculæ, which often persist after treatment, may require optical iridectomy at a later period. This, however, should be deferred as long as possible, since even an originally ectatic and very dense corneal opacity may be considerably cleared up after several years. I have observed this in a staphylomatous ectasia which called for enucleation of the globe. This is more likely to happen if tincture of iodine is applied often and long enough to the conjunctival limbus. A small, fine painter's brush is used, and so-called irritants are avoided long enough to induce involution of the infiltrate. The mildest of these remedies is calomel. It is applied to the palpebral conjunctiva daily, or every other day, in the shape of the finest possible powder by means of a large, fine-haired brush.

Massage of the eye with white or yellow 1-2 per cent. precipitate ointment is a more energetic treatment. Other ointments, used in the same way, are gray ointment (ung. hydrarg. 1.0, lanolin, vaseline \bar{a} 2.0) and potassium iodide ointment (pot. iod. 1.0, sod. bicarb. 0.5, vaseline 10.0). A little of the ointment, the size of a small pea, is applied with a glass rod to the lower lid, which is lightly drawn away from the eye. While the lid is held closed, the rod is slowly withdrawn toward the outer canthus. The lid is then gently massaged, so as to distribute the ointment uniformly in the conjunctival sac. Before applying the irritant for the first time, it may be advisable to instil homatropine hydrobromate (3 per cent.), to prevent hyperæmia of the iris.

In massaging the eye, no great pressure must be exerted on the globe. The procedure should be carried out quickly, but so gently that there will be no pain. The lids are moved over the globe in a horizontal, radial, vertical, or circular direction. The procedure should occupy no more than half a minute or a minute, and its repetition every other day, or once or twice weekly, depends upon whether the irritation of the globe produced by it soon disappears.

In order to prevent decomposition of the ointments, which in the yellow ointment is indicated by a grayish discoloration, they are kept in little jars, impervious to light, and closed by an equally impervious lid. Holth recommends yellow oxide of mercury ointment 0.05–0.20, lanolin 10.0. Schanz prescribes yellow oxide ointment 0.1–0.2, lanolin 1.0, distilled water 1.0, vaselin. ad. 10.0. Simultaneous internal use of iodine is to be avoided with these ointments, or to be prescribed as detailed on pp. 143–144, owing to the mercury iodine or iodide which is liberated.

In a torpid course of the affection, atomizing of opium tincture or dionin is beneficial.

When the eye has arrived at complete rest, subconjunctival injections of 1–2–3–5 per cent. saline solutions, either alone or with cinnamic acid (hetol pur. synthetic.), will promote resorption and involution of the infiltrate.

The injection is made in the following manner: After careful irrigation of the conjunctival sac with mercury oxycyanate (1 : 2000) a 2–3 per cent. cocaine solution is instilled twice or three times at intervals of two or three minutes. While the lids are kept apart by an assistant, an oblique puncture is made into the lower outer part of the bulbar conjunctiva with a sterilized Pravaz syringe containing saline solution, and the piston is slowly advanced until the syringe is empty. This raises the bulbar conjunctiva in the shape of a cyst. After several days, when the irritation has subsided, the injection may be repeated at other places, as, for instance, in the interspace between the external and superior rectus.

All this, however, must not be done until the climax of the affection has been reached or exceeded, and after the iridocyclitic irritative symptoms (ciliary pain, etc.) have disappeared.

Indications of this stage are (1) disappearance within half an hour to an hour of the congestion and redness of the vessels, conjunctiva, and episclera, caused by the irritant; (2) the prompt and uniform dilatation of the pupil upon instillation of a mydriatic, except when posterior synechiæ have persisted.

Persistent elevation of intra-ocular pressure—which occurs from numerous posterior synechiæ, or in rare cases without them—and pupillary occlusion may require paracentesis of the anterior chamber or

iridectomy. Sometimes there is an exacerbation, especially in the inflammatory stage of the process, due to increased pathological changes.

For *treatment* of corneal tuberculosis, compare Uveal Tuberculosis, p. 246.

10. CORNEAL CHANGES IN LEPROSY

A cornea affected with leprosy frequently presents changes which are easily recognized. The eyeball has a peculiar yellowish-brown shimmer, which may be seen at some distance. The leprosy infiltrates and nodules are usually located at the temporal portion of the corneoscleral border or in symmetrical area of the cornea. Even though there is but one small nodule at the margin, there may be an opacity elsewhere in the cornea. In other cases a light fringe separates the nodule and the opacity. The nodules are rather superficial, have a yellow-red to whitish appearance, and usually originate in the episcleral tissue; while those situated deeper have a rather gray appearance and arise from the region of Schlemm's canal. They terminate either in resorption or disintegration with perforation of the cornea and subsequent phthisis bulbi. More rarely are seen "smooth corneal infiltrates" which develop from several isolated spots and as a rule occupy the outer segment of the cornea and the "tuberous" corneal infiltrate. The superficial epithelial infiltrations may extend rapidly, the deeper ones more slowly. The former may protrude to such an extent that closure of the lids is difficult. Very active vascularization points to uveal involvement. There has also been described a superficial opacity which starts from the upper temporal sector of the border, and extends along the entire corneal periphery, in the shape of an arcus senilis. It may also appear as pannus leprosy in the shape of small, gray-white, superficial or deep spots around the scleral border, which have a tendency to extend towards the corneal centre, but diminishes with proliferation of the conjunctival vessels.

Leprosy changes of the lids and conjunctiva may be followed by anæsthesia and xerosis of the cornea, a mild keratitis e lagophthalmo, keratitis neuroparalytica, traumatic keratitis with secondary iridocyclitis, usually located in the lower half of the cornea, which may develop as rapidly as serpiginous ulcer, pannus and corneal scars which resemble pterygium.

Treatment.—The nodules are destroyed by red heat. To prevent their spreading over the cornea, its clear part, lying in front of the nodule, has been ablated with varying results. Sometimes the pupillary area of the cornea remained free, or, as in advanced processes, more satisfactory vision has been reëstablished corresponding to the involved cornea, which usually is the inner segment of the cornea. Optical iridectomy is of use in some cases. It has also been suggested to wall off the lesion by encircling it by the galvanocautery, following this procedure by inspersations of iodoform.

11. KERATOCONUS

This affection is more frequently unilateral than bilateral. It usually develops during and after puberty, although it is not unknown in adult life. The impaired vision, which is caused by abnormal corneal curvature, together with the thinness and opacity of its central areas, can only rarely be improved by concave or cylindrical glasses.

The hyperbolic curvature of the cornea is distinctly visible when viewed laterally and by the considerable distortion of the keratoscopic picture. The pupillary deviation in unilateral keratoconus is, according to E. Pagenstecher, due solely to the difference in the radius of curvature of the two corneæ and the different depth of the anterior chambers. According to my experience, the statement that keratoconus is associated with opacities of the lens cannot be accepted as a generally applicable rule.

Rupture of the corneal cone may occur in consequence of trivial injuries.

Treatment is, as a rule, difficult and tedious. In the initial stage of the stationary form correction by glasses may be tried. Should ectasia increase, instillation of physostigmine-pilocarpine-morphine, and pressure bandage during the night, should be carried out for a long time. The patient should be continuously in the recumbent position. The keratoconus may be carefully cauterized with pure carbolic acid. I use galvanocautery, after the method of Elschmig, governing the treatment by optometric and ophthalmometric tests. The central or paracentral scab can be connected with the corneal border any desired number of times without causing noticeable blemish.

Any occupation which requires great acuity of vision should be avoided.

Fick's contact glass, which is capable of improving the vision, is, unfortunately, not tolerated by the eye for more than a few hours. A better instrument is the hydrodiascope, which Siegrist constructed by so modifying Czermak's orthoscope as to make the concave glass easily removable and replaceable, while in Löhnstein's model it is fixed and immovable.

As to megalocornea and globular cornea, compare congenital glaucoma, p. 33.

Fascicular opacity of the interpalpebral area of the cornea, with degenerative changes, follows destructive corneal processes, chronic iridochorioiditis, or phthisis bulbi. If the conjunctiva is chronically and painfully irritated, it may become necessary to remove the deposits of lime, or, if the eye is incurably blind, evisceration or enucleation of the globe may have to be resorted to. In some cases the opacity may yield to treatment. This happened in one of my cases, that of an eleven-year-old child, upon whom an iridectomy has been done. Ohm made successful subconjunctival injections of potassium iodide in a nine-year-old girl whose visual acuity had been reduced to a minimum by dense corneal opacities.

VIII. AFFECTIONS OF THE SCLERA

OWING to the transparency of the overlying conjunctiva, affections of the sclera located in the anterior segment of the globe can be easily recognized. This is particularly true in unevenness of the surface due to elevations, such as occur in episcleritis and scleritis, new growths, cysts, cystoid scars and ectasia of the sclera (Plate VIII, Fig. 1), or to depressions due to ulcers. Axial myopia causes flattening of the bulbar equator; atrophy and phthisis bulbi cause retraction; while an interruption of continuity is due to rupture and perforating wounds. Penetrating wounds usually extend concentrically toward the corneal border, frequently involving the entire sclera, so that parts of the uvea, retina, and vitreous protrude through the lips of the wound.

Panophthalmitis involving the sclera imparts to it a yellowish, pus-like color. In scars of which parts of the uveal have healed, in ectasia of the sclera, in scleritis or partial ruptures, the discoloration is bluish-black or gray. It must be remembered, in this connection, that a congenitally thin sclera is of a decidedly blue color.

In old hemorrhages, infiltrated uveal pigment and melanotic tumors of the sclera and uveal tract, the sclera is of a brown or brown-black color. In cases of deep congenital pigmentation the pigmentation is intensified at the insertion of vessels and nerves.

In making a differential diagnosis, it is important to observe the dilatation and tortuosity of the anterior ciliary vessels up to their scleral insertion, a condition often present in chronic inflammatory and secondary glaucoma. In a circumscribed form this often indicates the internal site of an intra-ocular tumor. If the affected vessel is an artery, it fills anteriorly from the equator when forcibly compressed by drawing the lid over it; if the vessel fills in an opposite direction, it is probably a vein. Injection of the complete or partial annular scleral zone surrounding the cornea has already been described as a sign of inflammation of the anterior segment of the globe. In episcleritis and scleritis, this zone is either bluish or a violet-red and the tissues are swollen.

A scleral injection with small or large maculæ, located between the corneal border and the equator, points to pathological processes in the non-vascular sclera. It is due to greater repletion of the superficial scleral vessels, and is distinguished by its deep violet color from pericorneal and subconjunctival injections, which may be present simultaneously. The discolored sclerotic and episcleritic zones only reach the corneal border in a few places, if at all.

The inflammatory focal affections of the anterior segment of the

sclera are either superficially situated between conjunctiva and sclera (episcleritis) or in the deeper layers (scleritis). Transition from one form into the other often occurs. The clinical picture is as follows: There is either no inflammatory reaction at all, or there are irritative symptoms such as ciliary pain, lachrymation, photophobia, etc. At some distance from the corneal margin immovable nodular, whitish-yellow foci are sometimes found at the insertion of a straight muscle, sharply demarcated around the region of one or more episcleral veins. According to the congestion of the conjunctival, episcleral, and scleral vessels, these eminences have first a light red and later a reddish-blue, bluish or violet tinge. The unaffected parts of the sclera may be of normal color. This injection is changed to a dirty gray or bluish-violet discoloration of the sclera in the course of a few weeks or months. This condition should not be mistaken for congenital melanosis of the sclera. In atrophy and ectasia of the sclera it may become permanent, but these cases are usually complicated with an insidious iridocyclitis with an opaque vitreous.

The cornea may also be secondarily involved, as in tuberculosis and syphilis. If it occurs along with a diffuse corneal infiltrate, which is rare, the usual process is as follows: a vascularized, tongue-shaped, grayish, grayish-white or white opacity advances from the scleral focus toward the centre of the cornea or extends beyond it. It undergoes either partial involution or becomes permanent (sclerotic keratitis).

Differential Diagnosis.—In phlyctenular conjunctivitis the conjunctiva is involved, while in episcleritis the conjunctiva covering the scleral or episcleral focus is not only intact, but is quite movable. Phlyctenules are usually situated near or in the corneal margin.

In scleritis the tarsal conjunctiva and fornices are pale. The initial stages of gumma, leprosy, and scleral tuberculosis which have often been mistaken as malignant growths may simulate scleritis or episcleritis.

The course and prognosis of all forms of scleritis and episcleritis (two very obstinate affections) depend upon etiology and treatment. Thus, syphilitic scleritis and episcleritis, when recognized early, yield more or less rapidly to mercurial and iodine treatment, and tuberculous episcleritis to tuberculin therapy. Neglected cases show a tendency to relapse, with the result that gradually the cornea and neighboring structures become involved. Simultaneous presence of uveitis, with deposits upon the membrane of Descemet, renders the prognosis unfavorable, owing to the opacity of the lens and vitreous and the development of secondary glaucoma.

Treatment.—An energetic attempt should be made in all cases to improve metabolism by means of soap friction. A teaspoonful of brown

PLATE VIII.



FIG. 1. Scleral staphyloma in intracocular tumor.



FIG. 2. Hamburger's tentative treatment of iritis.

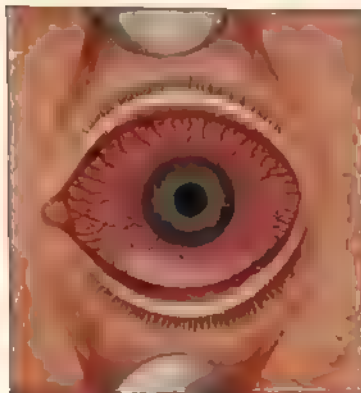


FIG. 3. Pericorneal conjunctival and subconjunctival injection. Iridodialis.



FIG. 4. Tubercle in the angle of the anterior chamber.



FIG. 5. Pseudoglioma.

soap is rubbed into various parts of the skin for five or ten minutes by the usual inunction method. Thermal and warm brine baths are likewise indicated. Tubercular processes are treated in accordance with the directions given on p. 246.

In syphilitic infections of anæmic and under-nourished children a strengthening diet is of greatest importance. If the disease runs a very prolonged course, mercury and iodine are beneficial. The same may be said of the use of certain waters, such as Heilbrunn, Kreuznach, Salzbrunn, Tölz, Hall (Upper Austria), Wiessee, and Wildegg, for both drinking and bathing. The continued use of Fowler's solution and arseniated waters (Dürkheimer, Maxquelle, etc.) often produces very gratifying results. Local measures include protection of the eyes by slightly smoked spectacles and total abstention from the use of the eyes, while tepid chamomile compresses, applied from fifteen to thirty minutes three or four times daily, or hot compresses and dry heat, often have a good effect.

Von Reuss applies the galvanic current with the Eulenburg electrode in the following manner:

After cocaine has been instilled in the eye, the electrode is grasped like a pen-holder and the polished side applied to the scleritic nodule, while the patient holds the second electrode in his hand. A 1-1.5 Ma. current is applied for from a minute to a minute and a half.

This time is distributed over whatever nodules are present. In sensitive patients, or in cases in which there is considerable reaction, the anode is first applied to the eye, and later the cathode. At first there is increased redness of the affected area. Coagulated mucus adhering to these places is often so slight that it can be wiped off, but if it should adhere more tenaciously, weaker currents must be used. This treatment is repeated on alternate days. As a rule, about twelve applications will decrease the subjective complaints rapidly and effect a reduction of the nodules. Some cases require more applications. Prolonged treatment is rarely necessary and failure quite exceptional. Relapses, however, cannot be prevented. In the presence of iritis or a rather diffuse process, which obscures the affected foci, direct galvanization is not tolerated so well as the faradic current applied through the closed lids. According to v. Reuss, the healing effect depends not only upon accelerated re-sorption of the inflammatory products as a result of the increased hyperæmia, but also on certain electrochemical effects; to wit, coagulation of the tissue at the anode as the result of acid production, and liquefaction of tissue at the cathode owing to the formation of bases.

Massage is most effective when given gently and for a short time with a 1-2 per cent. precipitate ointment on alternate days. This treatment often has a favorable effect upon the milder forms of scleritis and episcleritis. Its application, however, requires great care, as it may

lead to exacerbation in quite uncomplicated cases. It should not be applied in iridocyclitis.

If, in anterior scleritis or sclerotic keratitis, the corneal infiltrate extends rapidly, peritomy is advisable. Cl. du Bois Reymond has practised phlebotomy in a few cases with good results.

The technic is as follows:

The eyeball having been anæsthetized, an elevator is introduced between the lids. By means of a fixation forceps the eye is turned toward the healthy side, so that the affected area is freely visible. The principal trunks of the episcleral veins are raised with a small pointed hook, and cut through with a pair of scissors. When the hemorrhage has ceased, the eye is bandaged. If a neighboring area should become involved, the operation is repeated.

If scleral ectasia is imminent, the treatment consists, according to indications, either in the application of a pressure bandage with instillation of morphine, pilocarpine, and physostigmine, or iridectomy. The treatment of uveal affections, as explained on p. 234, may also be applied here.

Irritants are permissible only after the eye has become entirely quiet. This is especially true in sclerotic keratitis. The same applies to operations on the iris, which are also indicated on account of extensive corneal opacities.

Very small tuberculous nodules may be carefully scraped, provided there is no danger of thinning the sclera. Even a cure of large tuberculous scleral ulcers is within the range of possibilities, if competent treatment has been instituted early.

I observed such a case in 1890. There was an equatorial excavation of the globe with atrophy of the chorioid and sclera. The macular segment of the retina had been torn partly away from the layer of pigment epithelium, causing a reduction of central vision to six-twelfths of the normal, that of the other eye being six-fifths. With a bistoury I carefully dissected away the tuberculous focus at the margins as far as possible, and cauterized it in its entire extent with actual cautery. The resulting scleral defect was sutured with catgut and the latter reinforced by conjunctival sutures. The result was a smooth and permanent cure, with solid cicatrization of the sclera and central vision of six-sixths.

As to stretching of the sclera in the anterior and middle bulbar parts, it may be said that anterior and equatorial sclerectasia, as well as ciliary staphyloma, are usually caused by degenerative changes of the globe due to iridochorioiditis or glaucoma. They are often accompanied by pain of varying intensity. The globe being thin and enlarged, there is danger of rupture.

It is but rarely possible to preserve eyes of this description, even for their cosmetic effect. The attempt to do so involves a technically difficult iridectomy, gradual reduction of the staphylomatous protrusion of the sclera by multiple incisions, fixation of the ectasia by neighboring bulbar conjunctiva, and other similar methods.

In most cases, such as corneal staphyloma following blennorrhœa neonatorum, or conjunctival diphtheria, it is impossible to avoid ablation of the staphyloma and at the same time cover the sclera with conjunctiva. Enucleation is undoubtedly a better method. This should not be resorted to in children, because the growth of the orbit will be impeded, with consequent asymmetry of the face, and the conjunctival sac will continue to contract, in spite of the insertion of an artificial eye.

IX. DISEASES OF THE UVEAL TRACT

COLOR changes in the iris are of diagnostic significance. These changes may involve a part or whole of the membrane, and include localized hyperæmia and exudates of the stroma, or a complete change of color of the iris, as when blue becomes yellowish-green. Hemorrhages of the membrane occur, which leave behind, after absorption, a yellowish stain. When iron or steel particles are retained in the globe the iris becomes a brownish-yellow, due to deposits of oxide of iron (siderosis). In atrophy of the iris the tissue become thin, has a characteristic matted appearance, and becomes dark gray or of a dirty brown color.

New growths appear as yellowish-gray, orange-yellow, yellowish-brown, or dark brown spots.

These tissue disturbances also bring about changes in the iris marking. Similar changes in the iris pattern follow chronic infiltrations (tubercles, etc.), and the traction atrophy due to anterior synechiæ. Iritis without visible tubercles has often not been recognized as tuberculous until a diagnostic injection of tuberculin was followed by the rapid appearance of numerous tubercles in the iris and the neighboring sclera.

Displacement of the iris forward is caused by prolapse following perforation of a corneal ulcer, by impaction or entanglement in corneal and scleral scars and also by its being pushed forward by the lens. Shrinking of the vitreous, absence or retraction of the lens, causes backward displacement of the iris. Total adhesion of the pupillary margin to the anterior capsule of the lens produces a barrel-shaped protrusion, while irregular adhesions to the anterior capsule are followed by cystoid atrophy. Contraction of exudates at the posterior surface of the iris or an agglutination of the iris with the ciliary processes causes retraction of the periphery. Atrophy of the iris tissue associated with contracting exudates of the anterior surface of the iris leads to eversion (ectropion) of the pupillary margin, as is seen in chronic glaucoma. The ectropion may also be due to eversion of the pigment layer and encroachment upon the anterior layer of the iris.

Total iridonesis consists of distinct vibration, or an undulating tremor of the entire iris, upon movement of the globe. It is caused by absence or dislocation of the lens, general loosening of the zonula, or liquefaction of the vitreous. Iridonesis may not occur if the pupil is contracted, especially if coincident with anterior and posterior synechiæ. On the other hand, total paralysis of the iris, which is often present in traumatic displacement of the iris, increases iridonesis. Partial iridonesis is also occasioned by microphakia and subluxation of the lens, or a considerable tearing of the ciliary margin of the iris.

The superficial area of the iris is increased in hydrophthalmos and scleral ectasia. It is reduced in microphthalmos, phthisis, atrophy of the globe, in atrophy or rudimentary development of the iris, and in fissures and gaps of the iris (coloboma, diplocoria, polycoria, malformation of the pupils).

Irideremia, or complete absence of the iris, is either congenital or caused by grave injury or rupture of the globe. The iris, however, may still be present in the eye.

Rarely, as a consequence of iritis or injury to the iris, the anterior surface of the iris is flecked with fragments from the retinal pigment layer. Often there are lumps of pigment floating in the anterior chamber, or pigment scattered at its bottom. Deposits of pigment may be found on the posterior surface of the cornea, consisting of rust or hemosiderin following a hemorrhage into the anterior chamber.

The anterior chamber is often involved in affections of the iris and ciliary body, and it may be partially obliterated by adhesions between the iris and cornea. If the pupillary margin becomes totally adherent to the lens the rest of the iris bulges forward, making the anterior chamber shallow. New growths within or behind the iris, or in swelling of the lens in traumatic cataract, or advancement of the lens (glaucoma), produce the same effect. In dislocation of the lens different parts of the chamber are of different depths. In hypermetropia the reduced depth of the chamber is physiologic.

The chamber is deeper than normal in high degrees of myopia, keratoconus, corneal ectasia, and in certain types of cataracts. Shrinking of the vitreous, dislocation of the lens, loss of vitreous, and other inflammatory and traumatic conditions increase its depth.

The normally clear contents show pathological changes in suppurative keratitis, iritis, iridocyclitis, and iridochorioiditis in the form of diffuse turbidity, organized inflammatory, purulent exudates mixed with blood. Following rupture of the venous plexus of Schlemm or of the iris vessels and glaucomatous hemorrhages, blood may be found in the anterior chamber, while, following traumatic cataract, the swollen fragments of the lens occupy it.

The lower part of the chamber is involved in many cases (hypopyon, Plate VII, Fig. 3; hyphæma, Plate X, Fig. 5); while in others there are precipitates in the shape of punctiform, whitish, yellowish-gray or blackish exudates at or below the centre of the posterior surface of the cornea.

Special attention should be paid to the submiliary and miliary nodules in the ligamentum pectinatum and the ciliary part of the iris, or between the root of the iris and the cornea, which in parenchymatous keratitis and iridocyclitis can be recognized only by quite oblique illumination (Plate VIII, Fig. 4). With lateral illumination of the cornea

the relation of the nodules to the shadow of the limbus shows whether they do or do not touch the superior surface of the cornea. The ligamentum pectinatum forms the outermost wall of the anterior chamber, and, being impervious to light, throws a narrow falciform shadow from the limbus which falls upon that margin of the iris on the same side as the source of illumination. This is true also of foreign bodies in the sinus of the chamber.

The fact that the iris, ciliary body, and chorioid are so often involved in endogenous, metastatic bacterial and toxic infections of the eye, referable to congenital syphilis, tuberculosis, the acute contagious disease, intestinal auto-intoxication, etc., is largely due to the intertwined arrangement of their vessels and the narrowness of their capillaries. In spite of the most careful general and local examination, it is not always possible to establish the source of the infection or the starting point of the metastasis.

Either the anterior, medial, or posterior part of the uvea may be affected, while in purulent metastatic processes the entire uveal tract is involved. The inflammation may change from non-purulent into purulent iritis, cyclitis, and iridocyclitis. The tissue reaction is also influenced by the degree of sepsis of the capillary emboli occluding the vessels, and by the degree of disturbance of the general condition, of the state of nutrition of the eye, and of the uvea itself.

Nevertheless, there may be no visible changes in the uvea, and at first there may be no signs of its affection but hemorrhages or opacities of the vitreous.

In exceptional cases there may be a circumscribed affection of the ciliary body, which runs an abortive course and is characterized by a light, rosy, ciliary injection of a definite place in the ciliary region, by sensitiveness of the eye to pressure, and a feeling of pain upon accommodative effort.

This is often a prodromal sign of iridocyclitis, which in the very first stage is ushered in by a delicate, rosy circumcorneal injection (Plate X, Fig. 4), slight discoloration of the iris and often by fine punctate deposits in the lower quadrant of Descemet's membrane, by redness of the optic disc, and increased width and tortuosity of the neighboring retinal veins (so-called uveal hyperæmia).

Soon afterward there is increased turbidity of the aqueous, and in some cases a slight hypopyon and increased deposits upon the posterior surface of the cornea. Once I saw at an early stage a diffuse, perfectly uniform opacity of the lower and medial parts of the cornea, which disappeared as soon as the iritis was cured. Exudations in the pupillary space occur with contraction of the pupil and discoloration of the iris. Involvement of the ciliary body and the anterior section of the chorioid

may be accompanied by hemorrhages in the shape of fine specks or of filiform membranous flakes; and by pus-like, strongly reflecting opacities. This occurs, as a rule, in the anterior segment of the vitreous. At a later stage there may be whitish-yellow foci, which are themselves pigmented or surrounded by pigment, especially in the lower segments of the chorioid. Simultaneously there is increase of the pericorneal and ciliary injection, and varying impairment of the visual power. If the process runs a stormier course, there is often swelling of the upper lid, occasionally chemosis of the bulbar conjunctiva, associated with photophobia, lachrymation, and ciliary irritability. The latter gives rise to severe pain over the eyebrows and in the corresponding cranial hemisphere, and often to neuralgia of the teeth of the corresponding superior maxilla. When the ciliary body is involved the eye is sensitive to pressure. In the initial stage of iritis the tension of the eye is usually increased, but when the ciliary body is more severely involved, especially in contraction of exudates or blood effusions in the vitreous, the tension is considerably reduced. Involvement of the ciliary body and the anterior part of the chorioid often produces irritation of the ciliary muscle, a reduction of the distant point, as in myopia, and a general restriction of the visual field.

Hemorrhages in the anterior chamber have also been observed in iridocyclitis following hemorrhagic variola, and in Barlow's disease.

The *course* of the disease varies. There may be a complete cure, or permanent adhesion of the iris to the anterior capsule of the lens, pupillary occlusion, pupillary seclusion, and atrophy of the iris. Persistent opacity of the vitreous leads to opacity of all the layers of the lens. If the course is unfavorable, detachment of the retina and shrinking of the eye will occur, even after the lapse of several years. Blindness due to secondary glaucoma may be caused by extensive adhesions of the pupillary margin and the posterior surface of the iris to the anterior portion of the lens capsule. Complete loss of ciliary pigment has been observed in iridocyclitis, notably in sympathetic iritis.

The affection may last for many weeks, months, or years. It is an important fact for the prognosis and diagnosis that the central vision may be found normal, even in the presence of vitreous opacities.

On the other hand, the light sense may be considerably reduced. Consequently, there can be a complete cure only if the sensation of light has returned to normal and the "accommodative myopia" has completely disappeared.

Photophobia, lachrymation, pale red pericorneal injection, refractoriness of the contracted pupil to mydriatics, and sensitiveness of the eye to touch, may persist for a considerable time after the disease has run its course.

Differential Diagnosis.—The so-called pericorneal and ciliary injection, mentioned in connection with affections of the cornea, is of particular importance (Plate X, Fig. 4). It is due to congestion of the vascular tracts of the uvea, which anastomose with the venous plexus of the canal of Schlemm and the ciliary vessels of the episcleral and subconjunctival tissues. This sign is most pronounced in acute inflammations of the iris and ciliary body, less so in the subacute forms, and sometimes only slight or entirely absent in chronic tuberculous uveitis.

On the other hand, in very severe iridocyclitis, the conjunctival vascular area is more or less plethoric.

In order to distinguish between a superficial and an intra-ocular inflammation in doubtful cases, Hamburger is correct in recommending the administration of 2–4 Gm. of uranine in coffee. In from twenty to thirty minutes the conjunctival secretion of the healthy eye is green, the urine distinctly red, the skin lemon-yellow, and the sclera icteric, while the pupil remains black for hours. In inflammation of the iris, ciliary body, or cornea, however, coloring matter will pass into the aqueous humor after the same lapse of time. The color of the aqueous humor will then vary from the most delicate hues to saturated lustrous green, corresponding to the intensity of the inflammation (Plate VIII, Fig. 2).

The *prognosis* depends upon the nature of the primary affection, the character of the uveal affection, and timely, competent treatment.

Treatment is causal in all forms which depend upon endogenous sources. For this reason, hospital treatment is advisable even in apparently light cases, as ambulatory treatment, even if successful, requires a much longer time.

All use of the eyes must be avoided. They should be protected from bright lights by either moderately dark glasses or instillation of aq. zeozoni (see pp. 131, 132). Mydriatics are indicated if there is no increase of intra-ocular tension. They should only be used to prevent or remove posterior synechiæ so far as may be necessary, as, for example, in so-called serous iridocyclitis, which is usually of a tubercular origin, and then they had better be applied tentatively. Old adhesions will not be influenced by mydriatics. Atropine conjunctivitis is best prevented by keeping the solution pure and the dropper clean. Mydriatics are more efficient, as a rule, if applied in the forms of oils or ointments.

To prevent atropine poisoning, the patient is directed to close his eyes gently for a few minutes. During this time the eyes are covered with absorbent gauze, which has been moistened with 3 per cent. boric acid. Slight digital compression of the lachrymal canals in the region of the lachrymal sac, or compression of the nostril, with the head slightly inclined forward, is useful. If dilatation of the pupil has once been attained, it is sufficient to keep it so with a minimum quantity of atropine.

If mydriasis does not occur, the mydriatic is discontinued and a specialist called in. This should also be done in the presence of iris bulging or an annular posterior synechia. However, immediate iridectomy may in certain cases be very injurious, since by aggravating the inflammation it is liable to cause occlusion of the pupil in an already irritated eye. This holds good also for secondary glaucoma, which is not rare in serous iridocyclitis with a stormy onset. On the other hand, persistent tenderness and inflammation of an incurably blind or very soft eye may call for enucleation or evisceration of the globe.

If there is violent pain over the eyebrows and in the head, or if, as in cyclitis, even a light touch through the closed lids causes great pain, local bleeding by the application of one or two leeches to the temporal region (one or two fingers' width away from the external canthus) will often effect rapid alleviation of the ciliary neurosis, a distinct decrease of photophobia and a visible reduction of the turgescence of the iris, bulbar conjunctiva, and sclera. Of course, this procedure is not advisable in illy-nourished patients.

Warm cataplasms and other methods of heat application will do good service in conducting the blood away from the anterior segment of the uvea. Tepid compresses of borated water are also to be considered. The patient himself is the best judge as to which particular method is most acceptable to him and how long it should be continued. As a rule, the cataplasms are applied for half an hour to an hour, three or four times daily, or longer. Ciliary pains which become worse in the evening, especially after midnight, can often be favorably influenced by a hot foot-bath, to be taken for five or ten minutes immediately before retiring.

The patient sits on the ledge of the bed, warmly clad, and puts his feet into a high tub half full of hot water. At appropriate intervals more water at increasing temperatures is added until the skin of the feet, well above the ankles, is decidedly hyperæmic. The feet are then placed in cold water for a moment, and then briskly dried with a friction towel. The depletant action is increased if a hot-water bag covered with woollen cloths is placed against the soles of the feet during the night. A similar end is gained by hand-, forearm- and elbow-baths. The effect is increased by a simultaneous hand- and foot-bath.

As to internal remedies, owing to their diaphoretic action, only the salicylic preparations are to be considered (sodium salicyl., aspirin, novaspirin, etc.).

The dietary is of importance. Hot and alcoholic beverages, highly-spiced food or food difficult to digest, should be avoided. Constipation is best relieved, unless contra-indicated by the primary affection, by administration of sodium sulphate with powdered licorice root āā 15.0,

fennel oil gtt. v, $\frac{1}{2}$ –2 knife-pointfuls, according to age, to be taken in wafers before retiring. Gentler laxatives may be ordered.

In weakly or feverish children the skin function must not be unduly incited in an endeavor to hasten the absorption of vitreous hemorrhages and opacities. If the condition of the body is good, warm baths, carefully given, with subsequent packing in warm blankets, are of service. Perspiration may be hastened by "dialysé sudorifique Golaz" (5–10–15 drops in hot tea), or hot milk. A milder, though less effective, remedy is packing in well-wrung-out cloths which have been immersed in hot water. I deprecate pilocarpine. No doubt it acts more quickly, but it may cause vomiting and serious cardiac weakness.

Steam-baths are not appropriate in inflammatory affections of the eye, because they are liable to cause congestion of the head.

It should also be remembered that after long-continued diaphoretic treatment children are very liable to "catch cold." This is prevented by bathing them with tepid water and properly drying them after each treatment, and keeping them in the room for a while.

These procedures are best instituted one or two hours before or three or four hours after the principal meal, but never upon a full stomach. The diaphoresis over, the children are given a refreshing draught in the shape of a glass of milk, which, however, should not be gulped down rapidly, or a glass of fruit juice, such as strawberry or lemonade slightly sweetened with cane sugar.

Subconjunctival injections, which have been recommended in chronic uveitis for purposes of resorption, must be left to the decision of the ophthalmologist. They are useless when the affection has already led to shrinking and atrophy of the uveal tissue.

To prevent relapses, it is important in all inflammatory uveal affections that after convalescence the patient should not be exposed to glaring light or sudden changes of temperature, and that proper clothing should be worn to prevent cold in the lower extremities. Fatiguing eye work should be forbidden.

1. SYPHILITIC AFFECTIONS

Nearly all cases of endogenous iridocyclitis occurring in early life, as well as the typical parenchymatous keratitis which is often associated with it, are due to syphilis.

It may be imparted to the foetus during intra-uterine life, so that posterior synechiæ, pupillary exudates, atrophy of the iris, or shrinking of the globe are present at birth. Iridocyclitis, even without deep corneal infiltration, occurs in congenital syphilis more frequently in early than in late youth; and in the new-born is so strikingly free from irritation as to be pathognomonic. In syphilitic iridocyclitis appearing

later, the whitish-yellow or yellowish-red pupillary exudates may be of such extent as to cause pupillary occlusion. In other cases posterior synechiæ are few, the turbidity of the aqueous humor slight, although whitish punctiform deposits are found upon the endothelium of Descemet's membrane, along with involvement of the deeper ocular structures. And there are evidences of glaucoma.

Gumma of the iris, in the shape of a gray-yellow nodule, is rare in congenital syphilis. It is nearly always located in the ciliary portion of the iris, often spreads to the ciliary body, disintegrating into caseous or fatty masses. It leaves behind a more or less extensive atrophy of the iris, or a grayish-white scar which is usually quite adherent to the capsule of the lens. Occlusion and seclusion of the pupil, cataract, and extension of the gumma to the lens have been observed.

As the growth increases, it may fill the entire anterior chamber and lead to perforation of the sclera.

Differential diagnosis between gumma and conglomerated tubercles of the iris is not always easy. Usually, however, gumma is accompanied by miliary nodules. In unpigmented sarcoma, which is exceedingly rare in childhood, there are new-formed vessels, which are almost entirely absent in gumma as well as in conglomerated tubercles. Inflammatory symptoms occur earlier in gumma than in sarcoma.

Papulous iritis usually appears in the pupillary zone with strongly vascularized nodules. The uninvolved tissue is less infiltrated than in purulent iritis, and hypopyon is but rarely present. Tuberculous and leprous nodules of the iris appear as transparent gray or gray-white spots and are usually present in large numbers. The picture is a changing one—older nodules disappear and new ones crop up in other places, while in papulous iritis nodules occur only once and in limited numbers.

As to "normal iris nodules," compare p. 50.

Opacity of the aqueous humor, coarse deposits on the posterior surface of the cornea, hypopyon or hyphæma, point to inflammatory affections of the ciliary body and the chorioid. Frequent sequelæ are total or nearly total posterior synechiæ, calcareous cataract, fluid vitreous and detachment of the retina, an extension of the process to the sclera, with shrinking of the anterior bulbar segment. Total blindness sometimes follows. A more or less extensive exudate at a circumscribed area in the iris and episclera may simulate a new growth, gumma, or scleritis. Sympathetic involvement of the other eye may also occur in these cases.

The favorable and rapid result of mercury treatment in a number of cases seems to show that papulous changes may occur in the ciliary body as well as in the iris. In a few cases there was either a complete cure, or a cure with only a slate-gray or gray-blue area of sclerotic dis-

coloration remaining. The remnants of iridochorioiditis, such as deposits of uveal pigment on the anterior surface of the lens, posterior synechiæ, etc., may be a valuable help in the diagnosis of syphilis, particularly when neither the history nor the general findings justify a definite opinion.

The changes in the ocular fundus due to hereditary syphilitic chorioiditis are for the same reason of diagnostic importance.

The more subtle tissue-changes can be best recognized by stereoscopic, reflex-free ophthalmoscopy after Gullstrand, as it furnishes a larger and more detailed picture of the optic disc, retina, and chorioid than the vertical image.

Aside from inflammations of the chorioid which accompany or follow kerato-iridocyclitic processes, various forms of chorioiditis or chorioretinitis, with or without parenchymatous keratitis, occur.

Diffuse chorioiditis is characterized by a fine, dust-like, rarely floccular, actively motile opacity of the entire vitreous. To differentiate this condition, it is important to notice the early involvement of the external retinal layer, which manifests itself as a disturbance of the light sense in the shape of hemeralopia. This condition is often misinterpreted by the inexperienced as optic neuritis.

According to Sidler-Huguenin, there are the following types of hereditary syphilitic changes:

TYPE I.—A finely spotted, reddish-yellow flecking of the chorioid, the foci being from one-twelfth to one-twentieth the size of a papilla, with minute pigment specks which usually begin at the posterior pole, become more pronounced toward the periphery, or are present only in quadrants, often leaving the peripapillary zone and macula free. This condition is apparently the result of proliferation and atrophy of the pigment epithelium. The rest of the fundus is somewhat more pigmented. The optic disc is normal or slightly pale, and not atrophic. The retinal vessels are normal or slightly constricted. The visual acuity is normal, or at least still serviceable, the field of vision restricted, the light sense normal, the vitreous perfectly clear, and the pigmentation just barely recognizable in the inverted image.

In some cases there is a more distinct, finely-dotted, reddish-yellow or yellowish-gray mottling, and the pigment spots, which begin in the peripapillary region, are disseminated over the entire fundus, become more intense toward the periphery, and do not spare the macular region. The periphery of the fundus is usually gray or lead-colored, the nerve-head is pale or slightly atrophied. The retinal vessels are correspondingly constricted, but the vessel walls are rarely visibly affected. Central vision is impaired, being usually more markedly reduced on one side. The small, delicate, reddish or yellowish-red spots are in size from one-

eighth to one-twelfth of a papilla, and of circular outline. The pigment spots are nearly always round, seldom serrated like bone-corpuscles. The larger spots are the result of confluence.

The prognosis for this type, which is not closely associated with diffuse interstitial keratitis, is favorable, provided no complications like relapsing parenchymatous keratitis or mixed forms of the other types develop.

TYPE II.—Peripheral, round or oval pigment foci in the retina, isolated or in groups, which very probably originate from disease of the chorioid. The solitary, independent foci are usually from one-half to one-eighth the size of the disc. Fusion may result in extensive pigment plaques as large as several disc diameters. The single foci can still be recognized, at a later period, by the globular shape of the margins. There are also yellowish or reddish-yellow foci, which are nearly always round. The dark as well as the light foci may gradually extend toward the equator and posterior pole. The light foci are usually fewer in number than the pigment spots, show less tendency to fusion, and rarely spread to the chorioid. Chorioiditic spots are equally rare. Should they occur, they may undergo secondary pigmentation and finally assume disc-shaped figures.

The vitreous is involved only in the beginning, while in the end only minute and translucent opacities remain. The light sense and field of vision are but little changed as a rule. Reduction of the light sense indicates that there must have been some cause other than the affection of the fundus. A restriction of the field of vision, if present, corresponds with the advance of the affection from the periphery toward the posterior pole. The retinal vessels are rarely perceptibly affected. In slightly pigmented areas, however, or in chorioidal atrophy the chorioidal vessels are sclerosed. In very severe affections the optic nerve may undergo secondary atrophy, either in sectors or altogether, usually accompanied by contraction of the retinal vessels. The remainder of the ocular fundus is usually unchanged, except for the occasional presence of areas between the foci which are somewhat less pigmented. Still more rarely the fundus exhibits light connective-tissue striæ, caused by a red-gray discoloration between the dark foci. By far the most numerous and important changes are found downward, outward and downward, temporally, and inward and downward.

The affection occurs much more frequently in both eyes than in one, and is usually confined to the periphery. When dark foci occur at the equator or posterior pole, similar ones will be found in large numbers at the periphery. The reddish-yellow as well as the dark spots seem to be pushed forward, as it were, from the periphery toward the centre. In very severe cases the posterior pole and equator are just as strongly

attacked as the periphery; nor is the periphery exclusively attacked, when there is only a limited number of chorioiditic foci. In this type atrophic spots with visible chorioidal vessels, such as are often seen in disseminated chorioiditis, are exceedingly rare. The latter is usually preceded by diffuse interstitial keratitis with iritis. It is rare for the reverse order to be accidentally observed.

TYPE III.—At the onset of the disease there are roundish foci in the extreme periphery, which are at first reddish-yellow, but which rapidly become lighter. At first they seem to be located far away, perhaps still in the retina; later they rapidly enlarge and soon spread to the chorioid. Not all the small foci increase, but all assume a lighter gray or lustrous white color. Those located nearest the periphery usually fuse first, forming large atrophic areas which extend from the periphery in the shape of a light seam. From these more or less connected white, atrophic areas a number of foci, one behind the other, advance rather radially toward the posterior pole. These areas always go through the same process; they fuse rapidly, forming cones which bud forth like excrescences or projections, from the more peripheral, larger atrophied areas. Instead of forming a confluent light border zone in the periphery, the single foci occasionally fuse into smaller masses, which may assume any kind of configuration. Their margin forms large or small arches, according to whether the single foci were large or small. Centrally in front of the larger foci, there are usually groups of fresher solitary spots, either side by side or one behind the other.

The light foci are sometimes found quite unchanged after many years; in other cases they undergo pigmentation after a few months, which commences at the periphery. These changes depend entirely upon the primary affection. Consequently, if the retina and chorioid have been completely destroyed within a large atrophic mass, there is but little or no pigmentation to be expected in the interior of that focus. Those foci located in normal or slightly degenerated tissue will be more pigmented than the atrophic areas. The pigmented branches may form a network between the various foci. Or, again, instead of fusing they may only encircle the various foci marginally, or fill them up to a greater or less extent. This is the way in which the target figures originate.

With the advent of the secondary pigmentation the inflammatory process has run its course. If this takes place early, a diffuse interstitial keratitis will probably occur. If, however, the stage of secondary pigmentation is already present, it has usually been preceded by parenchymatous keratitis with iritis. At the onset, the vitreous is often seriously affected, especially at the periphery, but gradually it clears up, leaving behind a few small flakes or shreds.

Type II occurs bilaterally in most cases, but there are exceptions. As a rule, one eye is more involved than the other.

The prognosis in Types II and III is good, because there is no tendency to repeated exacerbations and extension of the disease. The parts of the fundus primarily involved remain normal; the optic nerve and retinal vessels do not become involved. Although visual acuity could be normal so far as the fundus affection is concerned, it is usually affected by complicating corneal opacities or spots in the macular region. The light sense is nearly always normal. The restriction of the field of vision is usually limited to the periphery, and depends entirely upon the extent to which the fundus changes have advanced toward the posterior pole.

TYPE IV.—This type is sharply distinguished from II and III, and very probably represents a general secondary pigment degeneration. There is increased pigmentation of the peripapillary zone, while the region of the posterior pole loses the normal red color of the fundus. The posterior pole assumes a uniform dark gray to ash or lead-colored, dull appearance, and is devoid of reflexes. The coloration is darkest around the optic nerve, changing to a lighter gray toward the equator. There are but few chorioidal vessels in the peripapillary zone, and the retinal vessels stand out indistinctly against the fundus. On the other side of the equator the dark coloration again changes to light red, from which it may be assumed that the pigment epithelium has disappeared. This part of the fundus is but slightly pigmented, or the blue-gray discoloration extends to the extreme periphery. The atrophy of the pigment, however, may occur only in spots between the larger pigmented plaques.

The fundus in this condition exhibits spotted pigment proliferation, which is more frequently round or oval in shape than square, stellate, or of the bone-corpuscle form. There is great variation in size, ranging from the finest pigment striæ and specks to plaques the size of one, two, three, or more disc diameters. The polygonal figures have fine branches, which anastomose with those of neighboring foci. They do not seem to be connected with the retinal vessels.

There also occur reddish-yellow, chorio-retinitic and pronounced light chorioiditic foci (Plate XVII, Fig. 1). Most of them are circular, in size from one-fifth to one-half or to disc diameters, and have no tendency to fuse with neighboring foci. They are rather isolated, and are usually confined to the equatorial zone or the periphery. They are considerably fewer in number than the pigment foci. Light spots are sometimes entirely absent, or very sparse.

The optic discs are sharply demarcated and pale, to a greater or less extent, and the retinal vessels correspondingly narrow. However, even in advanced cases, complete atrophy of the optic nerve is rare.

Affections of the retinal vessels occur oftener than in Types II and III.

Vision is nearly always bad, with diminished light sense and restriction of the field of vision, so that the prognosis is much more unfavorable than in the other three types. But it is better than in retinitis pigmentosa, because in the latter the light sense and field of vision decreases more rapidly with reduced illumination than in Type IV. Patients generally seek medical aid, owing to the hemeralopia, which appears at an early stage.

This type, like Type I, always occurs bilaterally, although one eye is usually more affected than the other. It is owing to the less affected eye that the functional disturbance is felt less.

Diffuse interstitial keratitis may also occur in this type, but, as in Type I, it is probable only as every individual suffering from hereditary syphilis is sooner or later likely to be attacked with parenchymatous keratitis.

All four types occur as "independent" affections of the fundus. They may be preceded by diffuse interstitial keratitis, but it is rare that the sequence is reversed. The peripheral changes of the fundus in Types II and III are in closer relation to that affection than Types I and IV.

Occasionally, there are mixed forms of various types in the same fundus.

Primary neuritis occurs relatively rarely in hereditary syphilis, while affections of the retina, which may be seen with the ophthalmoscope, are more frequent.

All these diseases generally run their course in the chorioidal layer containing the capillary vessels. The functional examination shows alternately decrease of accommodative range and paracentral visual defects, which gradually develop into very large annular scotomata. Aside from this characteristic symptom of fenestrated vision, photopsia and ciliary symptoms, there are sometimes complaints of micropsia and metamorphosia. Pronounced involvement of the macular region, especially in the presence of chorioidal hemorrhage, leads to a very obstinate scotoma with consequent marked impairment of visual power.

On the whole, with timely and competent treatment, the *prognosis* is not entirely unfavorable. But iridocyclitis, occurring in the first year of life, very often terminates with optic atrophy and extensive chorioretinitic changes, blindness and atrophy of the globe.

This is also true of gummatous iridocyclitis, which, fortunately, rarely attacks both eyes. In milder cases amblyopia persists, owing to the affection of the chorioid, retina, optic nerve and vitreous. Relapses may occur.

With the restrictions above explained, the prognosis of chorioid affections, generally speaking, is better if the affection is recognized early and competently treated. It is less favorable if slowly progressing syphilis is complicated by serious affections of the optic nerve and retina, hemorrhages in the fundus, cataract, and muscular paralysis.

The following case of H. Cohn shows how, in a markedly slow course of hereditary syphilis, affections of the eye may later occur, leading at once to partial or complete loss of sight or to processes extending over years:

At the age of eleven, a patient was seized with parenchymatous keratitis, nystagmus, and suppuration of the lachrymal sac. In spite of the most thorough mercurial treatment, chorioiditis set in several years later, followed by iritis. When twenty or twenty-one, there was glaucoma of both eyes. Iridectomy was then done. At the age of thirty-one, there was opacity of the lenses and with upward displacement. At thirty-three one eye was enucleated because of pain. At the age of thirty-seven, atrophy of the optic nerve occurred in the remaining eye, with the greatest possible impairment of visual acuity and night-blindness.

Treatment.—Mercury inunction (0.3–2.0 per day) is often effective. Part of the dose may be vigorously rubbed into the forehead and temples. The body weight and urine should be under constant observation. If the general condition is considerably affected by the treatment, or if even traces of albumen are repeatedly found, the treatment must be interrupted. This may occur before the onset of gingivitis. One-half to 1 per cent. hydrogen peroxide should be prescribed for a gargle, and not potassium chlorate, which has repeatedly caused poisoning in children who swallowed it.

If, for domestic reasons, inunction cannot be carried out the application of gray ointment to the forehead and temples is supplemented by sublimate pills. Sublimate baths are prescribed for children in the first years of life (1 Gm. to the bath). The duration of the treatment always depends upon the course of the disease.

Salvarsan has a strikingly favorable effect in the early manifestations of syphilis. In one of my cases an iridocyclitic hypopyon occupying half the chamber disappeared in a week after one injection. It is, therefore, worthy of consideration whether, in hereditary syphilitic eye affections of infants, the mother or a syphilitic nurse should not also be treated with salvarsan.

Injections should not exceed 0.1 Gm. and only be repeated several weeks later, if the first injection has been borne well. Careful clinical supervision is always advisable after meningitis. A nine-year-old girl, for instance, who had passed through luetic meningitis several years

previously, suffered on the fourth and fifth days several life-endangering attacks, lasting several hours, after only one injection of 0.2 Gm. salvarsan (Gilbert).

In congenital gumma, mercury is generally less efficacious. Better results may be expected from easily assimilable simple or compound iodines and arsenic preparations (iodferratose, arsenferratose, iodoglidin, sajodin, etc.). These remedies should be used, with temporary interruptions, for a long period; alternate preparations if indicated. The general health should also be built up. Occasional hydropathic treatment (wet packing, etc.) is often attended with good results. As to local treatment, comp. p. 234.

2. UVEAL TUBERCULOSIS

Miliary tuberculosis of the anterior uveal segment invades either the base of the iris, the ligamentum pectinatum, the canals of Schlemm, and rarely the sphincter area of the iris. The tubercles themselves in general follow the distribution of the arterioles, and less frequently the sphincter region. Usually the disease assumes the form of a chronic inflammation associated with deposits on the posterior corneal surface. The tubercles, which rarely exceed 1 mm. in size, as a rule are visible until late in the course of the disease. If these are very small and deep-seated, the clinical picture is often merely that of an acute or sub-acute iritis, with exudate in the pupillary region and opacity of the aqueous. When the tubercles show a tendency to purulent disintegration hypopyon may be present. While the prognosis is not absolutely bad in the milder forms, it is uncertain if the tubercles increase, coalesce, and form larger nodules. The prognosis is still worse in solitary tubercle of the iris. It is seated at the root of the iris and encroaches upon the cornea in the shape of isolated nodules or dense gray-yellow infiltrates, which in spite of vascularization may perforate at the limbus. However, this condition is rarely observed in infancy and childhood. There is often an uneven thickening of the iris with total adhesion of the pupillary margin to the anterior capsule of the lens, the so-called "butter ball iris." What has been said concerning prognosis in tubercular iritis applies to chronic tuberculous chorioiditis with scleral involvement. Granulomata of the chorioid also causes detachment of the retina, which later changes into the typical picture of the amaurotic cat's eye (Plate VIII, Fig. 5). This form of uveal tuberculosis leads with varying rapidity to purulent infiltration of the vitreous, to funnel-shaped detachment of the retina, and perforation of the sclera or to phthisis bulbi.

Differential Diagnosis.—The typical location of tubercle of the iris is in the pupillary region. In gumma of the ciliary body, which may become visible in the anterior chamber under conditions similar to the

solitary iris tubercle, corneal infiltration is chiefly restricted to the region of the focus. Its course is generally less malignant.

Miliary tubercles in the ciliary body often first betray themselves by recurring vitreous hemorrhages. Chronic chorioiditis, disseminated chorioretinitis, and inflammatory processes extending over the entire uvea, are often caused by tuberculous nodules and foci in the chorioid and retina.

Large nodular chorioidal tuberculosis exhibits large knots composed of a great number of small ones, around which the chorioid is infiltrated. It causes a diffuse and coarsely mottled discoloration, and a whitish appearance of the fundus, along with elevations in the retinal surface. Tuberculous neurorretinitis with considerable congestion of the neighboring veins and local retinal hemorrhages also occur. In addition are found, in the order named: iridocyclitis with vitreous opacities and pupillary exudates, steamy opacity of the cornea, conjunctival chemosis, and moderate swelling of the lids.

Typical chorioidal tubercles are seen with the ophthalmoscope as round spots of varying size, dull white or yellowish-white in the centre and with an increasingly red appearance toward the blurred margin. The centre may contain a dark pigment. In the larger spots, the course of the anterior retinal vessels and the parallactic displacement indicate a possible elevation of the retina. The smaller nodules may sometimes be better recognized, by direct examination, with strong illumination. Large chorioiditic exudates and grave chorioretinitis attended by extreme restriction of the visual field (resembling that of retinitis pigmentosa) do occur. As a rule, there is simultaneous retinal hyperæmia; often there are fresh hemorrhages and sometimes optic neuritis. There may, however, be no considerable disturbance of vision. These spots are differentiated from chorioiditic foci of a non-tuberculous nature by their quadrant-like arrangement, their indistinct demarcation, the pronounced round shape of the chorioidal nodules, the absence of pigment in the vicinity, and the distinct prominence of the larger nodules. Furthermore, the nodules are liable to grow, in which case they become whiter and more prominent in the centre, while fresh nodules of small size make their appearance.

In cases in which the *diagnosis* is doubtful an injection of old tuberculin is of importance. It causes a reaction with a steeply ascending and descending temperature curve. Its importance increases when the Wassermann test is negative, and when the injection is followed by increased ciliary injection, increased precipitates, and photophobia. Bernheimer recommends making the injection between the shoulder blades because, owing to their comparative immovability and their protected position, the injections will cause neither reactions nor pain.

According to Igersheimer, it is urgently necessary to control the visual power both before and after the injections. He observed a general reaction following injections of fractions of a milligramme, although neither the history nor the general examination furnished raised any suspicion of tuberculosis. In every case, therefore, all factors which point to a tubercular-ocular affection or to a latent general tuberculous infection must be weighed.

Chorioiditic foci of different structure may, however, furnish etiologically an analogical ophthalmic picture. In order to differentiate between retinal glioma and intra-ocular tuberculosis, Natanson called attention to the early spreading of the tuberculous process to the sclera, and the presence of inflammatory conditions.

Ophthalmoscopic examination of children in acute miliary tuberculosis is often so difficult that repeated observations may be necessary. The tubercles may not be visible until the affection has reached an advanced stage. Dilatation of the pupil for diagnostic purposes is required, because the tubercles may be located peripherally.

As signs of a healed tuberculosis of the anterior bulbar section, the following indications are important: Spotted or diffuse slight gray discolorations of the episclera near the corneal border, increase of the white condensed striæ in the conjunctiva and irregular extension of the corneal limbus, chalk-white discoloration of superficial corneal opacities, circumscribed roundish opacities and plethoric vessels in the lower and posterior corneal layers, anterior synechiæ in the sinus of the chamber, roundish defects in the anterior strata of the iris, and persistent chemosis without any other cause.

The local *prognosis* depends upon whether the tuberculous infection (which in acute tuberculosis of the iris often originates from tuberculosis of the bronchial glands or the lungs) is limited to the iris and ciliary body or the chorioid, or whether other parts of the eye are affected. In the latter case, termination in blindness with or without phthisis bulbi often occurs in spite of the most painstaking efforts.

Treatment always occupies a long time, usually six months. The general constitution must be strengthened and creosote preparations administered (guaiacol, carbon., histosan, etc.). The treatment should be supervised in a hospital by a specialist, particularly when anti-tuberculous serum Marmorek, original old tuberculin T. R., bacillus emulsion of tuberculin Béranek, are used. According to Sahli, the latter preparation is particularly commended, owing to the fact that it can be used in very small doses, and admits of very large variations in the quantity to be administered. Igersheimer found it very efficacious in such low doses as A/256 and A/512. According to my experience, the dosage for children should always commence with 1/10 c.c. A/512, because a

higher initial dose produces an increase of temperature, which is by no means to be ignored.

To prevent increased opacity of the vitreous and consequent increase of pressure, caused by the injection or its reaction in tuberculous iridocyclitis, it is necessary to commence with very small doses like 1/100 mg. T. The dose should be increased step by step, keeping the general condition and local reaction under constant observation. The latter should be watched with either the loupe or the corneal microscope, so that strong reactions may be avoided. This is even more urgent, if considerable fibrin and cell deposits are present on Descemet's membrane.

This treatment has so far been attended with the greatest success in affections of the iris and ciliary body with tubercles in the base or in the ciliary portion of the iris (Plate VIII, Fig. 4). It has been less efficacious in scleritis and parenchymatous keratitis, but gives better hopes in conjunctival tuberculosis, episcleritis, and disseminated tuberculous chorioiditis.

A. v. Hippel, like Bernheimer, prefers the bacillus emulsion, as being a better protection against relapses. He insists that the injections be continued until all the tubercles have been replaced by cicatricial tissue, the swelling as well as the vascularization of the iris has undergone involution, the precipitates at the posterior surface of the cornea and the vitreous opacities have disappeared, and the globe is entirely free from irritation. F. Schoeler recommends old tuberculin, because "it increases the receptivity and tolerance of the eyes for other remedies, and relapses are rarer without tuberculin." According to Sahli, tuberculin produces only a slight local hyperæmia and a slight increase of the exudation. Its irritative effect must therefore be watched lest the treatment becomes dangerous. It has also been advised that, as with inunctions, shorter periods of treatment be given, with certain intervals of interruption. Sometimes, in spite of a negative serodiagnosis, combined treatment with mercury, iodine, and tuberculin has effected cures.

I am rather partial to the application of soft soap, after Kapesser's method, by having it rubbed in over the abdomen or back every other day and allowing it to dry. It should be washed off within an hour if the skin is very sensitive.

I have also found that so-called serous iridocyclitis, running an acute or chronic course, is favorably influenced by inciting the salivary secretion with small doses of pilocarpine (R:—Pilocarpin. muriat. 0.2; acid. mur. gtt. xx; aq. dest. ad. 100.0; the doses increasing to 2–3–5 Gm.). It is to be taken in bed, where the patient should remain for two hours after the effect has passed away. I let patients take a teaspoonful of good wine before administering the remedy, in order to prevent unpleasant by-effects.

Local Treatment.—Like Pflueger and others, I have had good results from subconjunctival injections of hetol (comp. p. 222), and, provided there are no corneal infiltrates, from paracentesis of the anterior chamber. The latter procedure seems to act like the opening of the abdomen in peritoneal tuberculosis. It must be carried out very gently and may require general anæsthesia. According to conditions, I combine with this procedure loosening of the precipitates at the posterior surface of the cornea, as described by C. Schweigger, and, after the wound has healed, frequent instillations of pilocarpine-physostigmine-morphine (2 per cent., 1 per cent., 1/10 per cent.), alternating with dionin.

Hetol in 1-5 per cent. solution is perfectly harmless for the eye, even if applied for a long time, but as sodium cinnam. is chemically a very subtle salt, the preparation in the sterilized tubes should be used.

In tuberculosis of the iris and cornea, Koster has successfully applied air insufflations into the anterior chamber.

Early removal of the tubercles by dissecting out the affected parts of the iris is said to have been successful on various occasions, but it has led to dissemination of the tuberculous affection. Prophylactic enucleation has been recommended in grave cases which are no longer amenable to treatment.

On the other hand, Saemisch reports a case in which tuberculosis of the iris developed in the other eye after enucleation of an eye that could still count fingers at a distance of $3\frac{1}{2}$ to 7 feet.

The chance of a complete cure of uveal tuberculosis is increased by a prolonged stay in Alpine and sub-Alpine sanatoria, but tuberculous affections of the uveal tract have been cured without specific treatment, and mild cases have even been cured spontaneously. Nevertheless, there are plenty of cases which defy all treatment.

A typical tubercle of the iris, the size of a pea, in a child, disappeared almost completely after an eighteen months' stay in the country and administration of cod-liver oil (Königstein).

3. HEMERALOPIA

Functional disturbances caused by the various forms of chorioiditis and chorioretinitis are reduction of the central vision, retarded retinal adaptation in the shape of marked lowering of vision in reduced illumination (hemeralopia), central, equatorial and peripheral annular and retiform defects of the visual field, metamorphopsia and micropsia, the result of chorioretinitic exudates and scars which have effected a disturbance of the perceptive retinal elements.

Less constant are restriction of the borders of the visual field for white in daylight, paracentral scotoma, erythropsia and spasmodic accommodative spasm.

In neurasthenic and very sensitive children, fatigue of the field of vision may occur under the picture of Foerster's shift type. If the target of the perimeter is slowly shifted over the horizontal meridian from outward to inward until it disappears, and next over the same meridian from inward to outward, it will be found that the field of vision has become restricted. If, for example, the target was first recognized at 85° outward, it disappears in the reverse direction sooner—say at 80; or the limit where the target is recognized inward is likewise shifted—as from 60 to 40, etc. In this way there are two fields of vision, which overlap each other at the periphery, after the manner of a meniscus.

Hemeralopia in chorioretinitis is the result of acute malnutrition of the pigment epithelium, and disappears with the cure of the ocular affection. On the other hand, idiopathic congenital hemeralopia is an incurable complaint with acute exacerbations probably due to sclerosis of the chorioidal vessels, with degeneration of the outer retinal layers. Degeneration of the inner retinal layers and those of the optic nerve-fibres may follow. So-called recurrent hemeralopia, which recurs every spring for a shorter or longer period, is a transition form between acute and congenital chronic hemeralopia.

The hemeralopia which follows diffuse chorioiditis or chorioretinitis is, like idiopathic hemeralopia, characterized by moderate photophobia in bright lights, and abnormal dilatation of the pupil in the dark; by disturbance of the quantitative color sense in daylight (especially for blue), by disproportionate reduction of the visual acuity with reduced illumination and increase of lower sphere of irritation at Foerster's photometer. The limits of the visual field for white and colors narrow with increasing darkness. Blue disappears from the field of vision at a certain degree of darkness, although red is still perceived. In daylight, the field of vision for colors, especially blue, is constricted.

This so-called essential congenital hemeralopia is due to defective development of the retinal pigment. In chorioretinitis, hemeralopia is the result of local nutritional disturbances dependent upon exposure of the retina to intense light or to organic diseases which seriously impair general nutrition (*status hemeralopicus acquisitus*).

The central peripheral field for blue is of particular importance in exposing simulated night-blindness.

The *treatment* is etiologic or symptomatic. Light irritation may be relieved by keeping the patient in a darkened room or by the wearing of smoked glasses. General nutrition should be maintained with a full and easily assimilated diet. Pilocarpine is said to effect a striking acceleration in the secretion of the visual purple. Strychnine enlarges the color field for blue and red and temporarily increases the visual acuity

and amplifies the field of vision. Quinine and sodium salicylate may be given; phosphoretted cod-liver oil and "smoked" ox liver has the greatest effect, according to Changernier, Rampoldi, and Ligvies.

The wearing of smoke-gray spectacles is recommended for the photophobia due to albinism. Komos tried to improve the visual acuity of albinotic eyes by reducing the amount of light passing through the cornea, and by subconjunctival injections of India ink. Galtiero also tattooed the periphery of the cornea.

4. UVEAL AFFECTIONS IN LEPROSY

In addition to the iridocyclitis following corneal opacity, changes occur in the iris and ciliary body which produce a picture similar to tuberculosis of the iris. The simultaneous presence of leprosy and tuberculosis in the eye has been observed.

Milk-white, gray or gray-yellow nodules are found at the ciliary border, most frequently in the lower half of the iris. They may disappear spontaneously, to be suddenly replaced by fresh ones. The disease may involve the anterior chamber, attack the lower half of the cornea, invade the conjunctiva, and eventually lead to phthisis bulbi. According to L. Borthen, a typical sign of the dangerous *neoplasma lepromatosum anterius*, which often attacks cornea, iris, and ciliary body, is a discoloration of the episclera of the limbus, which begins with a more or less complete circular infiltration.

The slightest changes of a specific and non-specific nature are usually found in the maculo anæsthetic form of opacity. Upon ophthalmoscopic examination, the chorioid or retina has almost never been found to be involved.

Grave secondary phenomena are: Plastic iridocyclitis, chronic degenerative uveoscleritis, pupillary occlusion and seclusion, secondary glaucoma, opacities of the lens and chorioid, detachment of the retina, and shrinking of the globe.

Treatment.—According to L. Borthen, the light forms of iritis very often disappear spontaneously without the use of atropine. In truly leprosy iridocyclitis, however, atropine has no effect. According to the same author, cyanate of mercury 0.05, aq. dest. 50.0, sodium chloride 0.5, acin. 0.025, caused temporary arrest of the lepromatous eruption, with normal appearance of the affected area and increase of the visual acuity. One-half or a whole syringeful of the remedy is injected subconjunctivally between the cornea and the insertion of the muscles about every tenth day. Removal of the pathological focus by dissecting out the affected section of the iris is also said to have been successful. In some cases, however, this operation was followed by atrophy of the globe. At any rate, iridectomy might have to be considered in persistent

increase of pressure due to grave iridocyclitis, and enucleation of the atrophic globe became painful.

5. RECURRING VITREOUS HEMORRHAGE

Recurring hemorrhages of the vitreous originate most frequently in the vessels of the ciliary processes, although sometimes in those of the chorioid or retina. These hemorrhages are often followed by proliferating retinitis. The disease is more frequent in boys between ten and fifteen years of age, or at puberty, and is often due to a hereditary syphilitic or tuberculous affection of the ciliary body. They have also been interpreted as a phenomenon, analogous to epistaxis, which also occurs at puberty, or as an indication of a hemorrhagic diathesis. Diffuse or coarse, clumped opacities of the vitreous occur with insidious uveitis—that is to say, without noteworthy inflammatory changes of the iris and chorioid. The hemorrhages occur in one or both eyes, often involving the entire vitreous, and causing a sudden reduction of the visual power. Vision in this instance is reduced to the ability to count fingers directly before the eyes or even to mere quantitative perception of light. Resorption takes place only after a considerable time. Should the hemorrhages not be resorbed, there will be considerable lowering of vision. Detachment of the retina has been observed as a consequence of connective-tissue degeneration of the vitreous. In the more favorable cases, after the vitreous has cleared up, there are often found large or small, irregularly-formed, yellowish or reddish-white peripheral chorioid and retinal foci, which are partly or wholly encircled by pigment.

The *treatment* is generally etiologic, and its results are more favorable, if commenced early.

Elschnig suggests substituting the opaque vitreous flakes, aspirated by the Pravaz syringe, by a 0.85 saline solution, but he does not substitute for more than 0.6 c.c. of the vitreous. Further reports on this procedure are desirable.

An ophthalmologist should always be consulted for the treatment as well as the diagnosis.

In the differential diagnosis, foreign bodies and parasites in the vitreous (*cysticercus cellulosæ*, *filaria*) must always be considered. The difficulties of diagnosis can, in general, be increased by the presence of diffuse opacities of the vitreous. In a nine-year-old child, tubercular chorioiditis was diagnosed on the strength of the history. It was only after the fundus had cleared up by internal application of iodine and subconjunctival injections of a 4 per cent. salt solution that a correct diagnosis could be made, and a *cysticercus* removed by operation.

Hemorrhagic affections of the chorioid are also to be considered. They occur in early life, after grave dysentery, and usually defy all

treatment. In one case I observed ever-recurring hemorrhages, with progressive atrophy of the chorioid and the posterior retinal layer, principally of the macular region. This led to a much reduced vision, and finally to total blindness.

6. ACUTE METASTATIC SUPPURATIVE IRIDOCHORIOIDITIS (PANOPHTHALMITIS)

This disease follows injuries and operations, umbilical inflammations of the new-born, pulmonary empyema, phlebitis, diphtheria, mumps, acute exanthemas (scarlet fever, measles, varicellæ, variola, etc.), acute inflammations of the face, and occasionally miliary tuberculosis.

The clinical picture varies. In unilateral metastatic suppurative uveitis all parts of the eye are attacked indiscriminately. In bilateral metastatic ophthalmia the development of capillary emboli is favored by the arterial system of the retina, with its terminal branches. Some cases set in with only a pale chemosis of the bulbar conjunctiva and hypopyon. In others there is redness of the conjunctiva, with more or less pronounced cedematous swelling of the palpebral margins, associated with swelling of the bulbar conjunctiva and episclera, in such a way that the cornea is encircled, as if by a rampart. The cornea may be transparent, hazy, or diffusely striated, or, in grave cases, near the periphery the middle layers are infiltrated in an annular form, with yellowish pus. Simultaneously, there appear hypopyon, discoloration and swelling of the iris, and yellowish exudates in the pupil, which is of medium width and active. If the cornea, anterior chamber, and lens are still moderately transparent, there is often a yellowish shimmer coming from the depth of the eye, due to exudates behind the lens. These changes are accompanied by pains in the eye and vicinity, because of the tearing of the sensitive nerve branches. Pains are often piercing at first, and, later, even more violent. There may be vomiting and fever at the onset of the process. In general cachexia, however, these symptoms may be absent.

The reduction of visual power, which is perceptible to the patient, rapidly develops into blindness.

The swelling of the lids and the pains increase, the yellow marginal ring spreads over the entire cornea, the bulbar capsule becomes distended, the globe protrudes, and the result is suppurative disintegration of the ocular contents and perforation of the intra-ocular abscess. This is followed by gradual shrinking of the globe and so-called phthisis bulbi. Meantime pain and the palpebral œdema have decreased.

It is rare that all the phases of this process can be followed with the ophthalmoscope, as it takes place in the interior of the eye. Just as rarely can the starting point of the metastasis be definitely established. However, when the metastasis occurs in the anterior segment of the

eye, the iritic symptoms are the most prominent, while at first the vitreous shows but a slight diffuse opacity.

If the metastasis is deeper and develops more slowly, examination, in the beginning, will show numerous flaky vitreous opacities, considerable distention of the retinal veins, numerous punctiform and linear retinal hemorrhages, striation, swelling, and yellowish-white discoloration of the retina at the original focus of the metastasis.

The lens, too, may show finely striated opacities, which subsequently spread over the entire structure, sometimes with great rapidity.

The *prognosis* of bilateral metastatic ophthalmitis, which sets in at approximately the same time in both eyes, is, with rare exceptions, bad. It also threatens life. On the other hand, in unilateral metastasis, children's lives have been saved. These metastases, originating by transportation through the circulation, are distinct from suppurative chorioiditis occurring in inflammation of the brain, or in and after epidemic cerebrospinal meningitis. These latter conditions are produced by the transportation of septic emboli through the ocular lymph-tracts. The course of the latter displays symptoms similar to those of arterio-embolic metastasis, but the cornea remains intact. The disease nearly always terminates without perforation of the globe but with pupillary occlusion and seclusion, opacity of the lens, shrinking of the vitreous, detachment of the retina, and shrinking of the cornea and the rest of the eye. It is extremely unusual in these conditions for even a part of the visual power to be restored.

Treatment.—In arterio-embolic chorioiditis, which runs a more or less rapid course, severe pains, if present, are often relieved by cold compresses. In other cases, moist or dry heat gives a more pleasant sensation, and at the same time accelerates the course of panophthalmia. It may also be necessary to administer a sudorific or anodyne.

Perforation is in most cases spontaneous; and only rarely must the globe be incised in order to evacuate the pus. It is also advisable to apply absorbent compresses, which are to be changed once or twice a day, and preceded by careful irrigation of the conjunctival sac with a freshly sterilized 3 per cent. boric acid solution.

An artificial eye will have to be worn later, just as after enucleation of the globe. It should be fitted well and kept in proper condition, as it is the only means of preventing chronic catarrhal irritation of the conjunctiva, contraction of the orbital cavity, and asymmetry of the face.

Before inserting the artificial eye, the lids and conjunctiva are cleansed with absorbent cotton which has been moistened with boiled 3 per cent. boric acid. The artificial eye is similarly treated, as it slips in better when moistened.

The way to insert the eye must be demonstrated to the parents by the physician (Fig. 33). There must be a good light. The child sits on a chair, or on the lap of his mother or nurse, and it may be necessary to hold his hands and feet, in the first few insertions. Assuming that a left prothesis is to be inserted, the physician, sitting opposite, slightly draws away the upper lid with the thumb and index-finger of the right hand. Directing the child to look downward, he takes the artificial eye between the thumb and index-finger of the left hand, with the short part directed toward the nasal side, and slips it upward and backward underneath the upper lid, until it touches the upper fornix of the conjunctival sac. While it is held in this position with the thumb and index-finger, the right hand lets go of the upper lid and gently pulls the lower one down-

FIG. 33.



Insertion of an artificial eye.

ward with the index-finger, so that the lower edge of the artificial eye may glide in behind. Should the position not be comfortable immediately, the lids are slightly pulled up and down with the thumb and index-finger.

To remove the prothesis (Fig. 34), a squint-hook is used (Plate XXI, Fig. 1), which must always be kept absolutely clean. The lower lid is drawn away with the left hand, and the hook inserted with the other by means of a slight twist behind the lower edge of the artificial eye, so that the point of the hook will touch its posterior surface. By slightly tilting the hook forward, the artificial eye appears between the lids and beyond the free border of the lower lid. As soon as it has come about half-way out it may be grasped with the thumb and index-finger of the disengaged hand. The insertion and removal of protheses, which, for cosmetic reasons, consist of thin shells to be applied over a blind eye,

demand great care and skill, in order to prevent undue irritation of the globe. This is especially true if sensation remains in the cornea. The insertion is facilitated by the patient looking strongly downward, and the removal by his looking strongly upward. Parents should be cautioned that these procedures must always be carried out with absolutely clean hands and short-trimmed nails. The eye is removed in the evening and carefully and gently cleaned with a toothbrush and soap. The water used should be clean and lukewarm so as to prevent cracking the eye, which in the course of the day has become warm. During the night it should lie on clean cotton in a small wooden box.

The prosthesis is at first worn only for a few hours at a time, but later, when the sensitive conjunctiva has become accustomed to it, it is worn all day. If mucous secretion continues the eye is removed and cleaned once or twice a day. From time to time it is washed with ether, so as to remove thoroughly all adherent and hardened particles. After the eye has been worn for a year or two, or even less, it will become somewhat rough, and should be replaced by a new one. Otherwise there will be chronic irritation or even proliferation of the conjunctiva, which, like swelling of the conjunctiva, results from want of cleanliness and may cause progressive shrinking of the conjunctival sac to such an extent that an artificial eye cannot be worn. The prosthesis, therefore, should not be worn unless it is perfectly smooth.

An artificial eye, in order to fit perfectly, should be of such size and shape as to just fill the orbit. It should be tolerably well movable, show the cornea in correct position, and not interfere with closure of the lids.

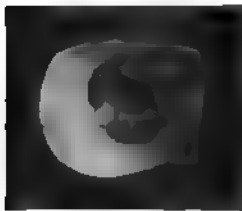
For this reason, the indiscriminate use of any prosthesis will not do. It should be specially made for each case. A double-walled "Reform Eye" (Fig. 35) has been devised by Snellen, which is in use, in addition to the ordinary shell-like form. The contour and capacity of the conjunctival sac differ considerably in individual cases, and must be duly considered. This is especially necessary if, because of symblepharon, etc., the structure of the conjunctival sac is irregular.

FIG. 34.



Removal of an artificial eye.

FIG. 35.



Improved ("Reform") eye.

The first insertion is effected over a closed and non-irritative conjunctival sac, and may, therefore, be done as early as two weeks after enucleation of non-inflamed eyes. In panophthalmia, complete healing of the perforation and abatement of all inflammatory signs of the conjunctival sac should be awaited. In exenteration of the globe, I defer the insertion until the traumatic cavity is well healed. In order to prevent retention of injurious matter, I do not suture the sclerotic, being content with a thorough cleansing of the scleral cavity with a $\frac{1}{2}$ per cent. solution of hydrogen peroxide. Phthisical eyes will tolerate prosthesis only if they are not sensitive to pressure.

If, in spite of all precautions, a pronounced catarrh of the conjunctiva should set in, the prosthesis should not be worn for more than a few hours at a time, or be discontinued for a time.

It may be necessary to instil a zinc solution, or to paint the tarsal conjunctiva with a $\frac{1}{2}$ -1 per cent. solution of silver nitrate.

Younger children who are doomed to blindness should, while there are still remnants of vision, be taught the typewriter or the blind alphabet, or be sent to an institution for the blind, so that the education of their sense of touch may have the advantage of what vision they have.

7. UVEAL TUMORS

Tumors of the uvea are rare in young persons. Melanosarcoma of the chorioid has been mistaken for conglomerate tuberculosis.

The *treatment* of uveal tumor is enucleation. As mentioned on p. 20, spontaneous cure of cyst of the iris is extremely rare. The methods usually practised, such as puncture of the cyst, with or without destruction of the wall, and radical extirpation rarely save the eye from loss of sight or total destruction. Enucleation is frequently necessary.

In one case, A. Schoeler caused the cyst to contract without recrudescence in four and a half years. He punctured the cyst with a Pravaz needle and, under slight pressure, injected, in one case diluted, in another case undiluted, tincture of iodine, drop by drop. As a result the cyst contracted without giving rise to very active irritation. "A partial opacity of the anterior capsule of the lens, caused by the iodine, can probably be avoided in future by using eserine before the operation and proceeding carefully."

For blood cysts in the ciliary body, see "Injuries."

X. AFFECTIONS OF THE LENS

If the lens is of normal size and in normal position, it is hidden from the view of the unaided eye. Even with a widely-dilated pupil and aided by lateral illumination, its periphery is hidden by the iris. The entire lens can be seen only in total irideremia, and in part from the centre of the pupil to the equator, in congenital traumatic or operative coloboma of the iris. The presence of the lens is demonstrated by the so-called Purkinje-Sanson pictures, which are formed by reflection of a source of light by the anterior and posterior surfaces of the lens.

In making a differential diagnosis the following conditions must be considered: Capsular cataract, deposits upon the posterior surface of the anterior lens capsule, and opacities in the substantia propria, which to the ophthalmoscope are either dark or impervious to light, and which appear as whitish spots only in reflected light. These opacities should not be confused with the deposits upon the anterior capsule of the lens known as spurious cataract, which are either pigmented residues of the pupillary membrane, or pupillary exudates containing blood-vessels or hemorrhagic exudates, connecting the pupillary border with the anterior capsule of the lens. So-called "posterior polar cataract" is either a residue of the hyaloid artery, or a deposit upon the posterior capsule from vitreous opacities.

Deposits of this description upon the anterior capsule of the lens may be associated with capsular cataract, or with opacities in the anterior lens layers.

The extent and position of an opacity of the lens can be recognized (1) by the width of the deep shadow of the iris, which gradually darkens from the pupillary margin towards its blurred border; (2) by the speed of the parallactic displacement of the opacity toward the pupillary margin; and, according to conditions, (3) by the shape of the opacity. They are quite flat or anteriorly convex in the anterior layers of the lens, and concave in the posterior layers.

If the adjacent parts of the vitreous are opaque, the reflex picture of the posterior surface of the lens is more distinct and brighter. If the opacity is in the lens, it disappears or becomes indistinct, opaque, yellowish-red or with red preponderating. Furthermore, the following details must be considered in opacities of the substantia propria: shape, marking, demarcation, density, lustre, color, arrangement of position, extension toward the surface and depth, and changes in the shape of the lens. The lens may be larger or smaller than normal. Its capsule may protrude, as in cataract of the anterior capsule or pyramid cataract, or it may be indented, as in incomplete resorption of traumatic cataract, or wrinkled. Lateral illumination or examination with transmitted light, and artificial dilatation

of the pupil, will show whether the capsule and the substantia propria are uninjured, or whether a foreign body has penetrated the lens.

Of practical importance in childhood, above all, are: Congenital conical bulging of the anterior or posterior surface of the lens, anterior and posterior lenticonus, and congenital coloboma of the lens with defective development of the zonula. In congenital coloboma the lower border of the lens is either flat or concave downward. If the lens is dislocated it may be either slightly or freely movable and part of its border may be visible in the normal or dilated pupil. Congenital dislocation or ectasia (Plate IX, Figs. 5 and 6) is caused by defective development of an elongation of the zonula on one side. It is usually associated with defective development of the lens (microphakia), and sometimes with coloboma of the lens, iris and chorioid, and with aniridia. Dislocation is also due to atrophy, tearing or pulling of the zonula by anterior ectasia of the sclerotic or adhesions of the lens to corneal staphyloma, or to erosions from pus foci.

Absence of the two reflex pictures, great depth of the chamber, tremor of the iris, a high degree of hypermetropia, and a deep black pupil point to a congenital or acquired dislocation. Dislocation, in turn, may be into the anterior chamber, pupil, or, if the sclera has been ruptured by traumatism, the lens may escape through the wound and lodge under the conjunctiva or vitreous. Operative removal of the entire lens presents the same picture of aphakia.

Absence of the capsular contents due to cataract operation (so-called spontaneous resorption), or tearing of the anterior or posterior capsular membrane, is often betrayed by a few persisting residues (secondary or contracted cataract).

Advancement of the lens occurs in secondary glaucoma, total posterior superficial synechiæ, atrophy of the iris and ciliary body. Retraction of the lens follows in loss of the vitreous, perforating wounds or shrinking of vitreous exudates, etc. Tremor of the lens occurs in relaxation or tears of the zonula, liquefaction of the vitreous and cataracta tremulans.

Among the congenital anomalies of the lens, mentioned on p. 23, the most frequent is laminated cataract (cat. perinuclearis, zonularis, Plate IX, Figs. 3 and 4), which always affects both eyes. The clear central area of the lens is surrounded by a layer of opaque lenticular substance, from which solitary punctiform opacities or radially arranged conical processes extend into the clear anterior and posterior cortical substance. The development of zonular cataract is often rudimentary, so that instead of a round, slightly diffuse or radial, sharply demarcated opaque disc around the centre of the lens, there are often but fragments of the latter or a number of opaque radii. In the rarer form of double or treble laminated cataract, the shell-like zonular opaque layers are

separated from each other, as well as from the nucleus and cortex of the lens, by transparent lenticular substance.

In pyramidal cataract, the centre of the anterior part of the capsule protrudes conically into the anterior chamber. It is a variety of anterior polar cataract and is nearly always due to perforation of a central gonorrhœal ulcer of the cornea.

Posterior polar cataract also occurs in the course of retinitis pigmentosa.

Soft total cataract was observed by v. Michel in a twenty-one-months-old child who was artificially fed. The child presented the picture of so-called *atrophia infantum*, due to dysentery and diarrhœa, the latter persisting for three months. The same author observed this affection associated with anterior annular capsular cataract in a moderately well-nourished child whose father was syphilitic; and also a bilateral contracted cataract with nystagmus, but without any signs of uveal affection, in a weakly, under-developed girl who had had cerebrospinal meningitis in the first year of life.

In regard to the cause of congenital hard nuclear or total cataract, and the form of total cataract occurring in children between four and twelve years of age, such as often occur in one or both eyes in the presence of hereditary syphilis, v. Michel suggested a local disturbance of circulation due to affections of the walls of the common carotid and its branches.

Diabetes in the young, which is happily rare, and phosphaturia, have also to be etiologic factors.

Calcification of the lens (calcareous cataract) is often accompanied by gradual atrophy of the globe, and is nearly always caused by grave, destructive uveitis.

In order to *diagnose* these changes of the lens, lateral illumination and examination with the ophthalmoscope are necessary. In all doubtful cases the patient should be referred at once to an ophthalmologist. This is also advisable if operation becomes necessary. Even the ophthalmic surgeon may find a contracted cataract technically more difficult, owing to thickening of the capsule, than a maturing or matured form. It is impossible to predict whether a non-stationary opacity will progress slowly or rapidly. Generally speaking, fine, speck-like opacities with dark, very fine and shallow interspaces point to slow growth, while broad, opaque sectors with wide interspaces, penetrating deeply into the substance, point to rapid growth or soft cataract.

It is important for the *prognosis* to know whether the perception of light is good. An otherwise normal eye with total cataract will, as a rule, still recognize rays of candle light in the dark at a distance of at least twenty feet. The light of a flame reflected into the eye by a plane mirror from above, below, etc., should be correctly localized. Correct recognition

of pieces of colored paper or cloth, about forty inches square, proves the absence of complications with glaucoma and retinochorioditis. But unusually strong condensation of the opaque lens may so impair the perception of light and the color sense that candle-rays will be recognized only at a nearer distance, while the perception of color is uncertain or absent.

The operative prognosis of calcareous cataract, and the total cataract designated as chorioidal cataract, is less favorable, partly owing to the visual impairment caused by the chorioiditis, and partly to other defects of development (microphthalmos, coloboma, aniridia, etc.).

FIG. 36.



Eye screen.

For cataract in heterochromia of the iris, compare p. 41, and for traumatogenic and traumatic displacements of the lens, see chapter on "Injuries."

Treatment.—The description given on p. 25 holds good here, too, in a general way. Cataract operations heal more easily by replacing the bandage, which in children cannot well be dispensed with for the first few days, and by using an eye screen (Fig. 36), to be worn until the eyes are past all danger.

Children with congenital cataract who have not regained their vision until between their fourth and seventh years will at first have difficulty in judging distance and the form and size of objects. For a long time they will therefore avail themselves of their sense of touch and learn only with increasing practice to locate an object accurately (comp. remarks made on p. 57).

PLATE X.



FIG. 1. Exophthalmos due to retrobulbar effusion owing to contusion.



FIG. 2. Foreign body on the conjunctiva of the upper lid.



FIG. 3. Laceration of the conjunctiva and cornea.



FIG. 4. Pericorneal ciliary injection. Corneal scar with adhesion to the iris.

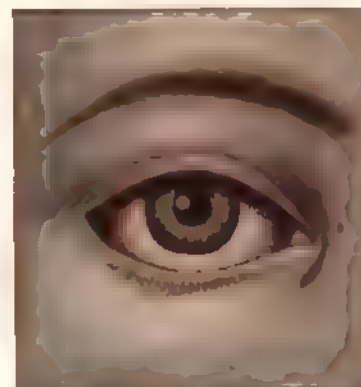


FIG. 5. Blood effusion into the anterior chamber in contusion of the bulb.

XI. GLAUCOMA

(PLATE XV, FIG. 1)

For congenital glaucoma, comp. p. 33; and for secondary glaucoma the remarks made in the chapters on Cornea, Sclera and Uveal Tract.

Although glaucoma is very rare in infancy and childhood, it is a possibility which must be kept in view. Elschnig reported the case of an infant with eclamptic paroxysms following gastric disturbance, whose eye reacted to the intoxication with an attack of glaucoma.

Differential Diagnosis.—The usual methods of determining ocular tension, or the intra-ocular pressure, consist in exerting from above a gentle pressure upon the globe, through the lightly closed lids, with the points of both index-fingers held near together. (The examining fingers should be warm.) In so doing, the tension of both eyes of the patient as well as the physician's own is compared. The examiner's arms are held in quiet equilibrium, without any muscular tension. But this test is very unreliable, and the pressure is measured (ophthalmotometry) more precisely with the Schiötz tonometer. The instrument should be kept scrupulously clean, work correctly, and be used in accordance with instructions. The eye should not be strongly irritated and the cornea not irregularly curved. With these premises, the results will be satisfactory in older children. Younger ones sometimes present insurmountable difficulties. The first step is to render the conjunctiva and cornea insensitive by alypin (2 per cent.). Cocaine and its chemically related substitutes are unsuitable, as they might injure the corneal epithelium. With this method it is possible to demonstrate positively every slight elevation of the intra-ocular pressure in an eye whose optic nerve has been excavated by glaucoma without showing externally signs of the disease. The average normal pressure in children is 18 mm. of mercury.

If the elevation of pressure has been rapid and pronounced, there will not only be distinct hardness, which can be demonstrated by palpation also, but often anæsthesia of the cornea. The cornea looks dull, like breath on glass, and is diffusely opaque, particularly in the centre parts, so that the condition may be confused with diffuse interstitial keratitis. The iris is discolored, the pupil opaque and irregularly dilated, and reacts sluggishly or not at all. The anterior ciliary arteries are greatly distended, the conjunctiva chemotic and hyperæmic, and the lids œdematous. The capacity for counting fingers or recognizing the hand close before the eyes is either reduced or completely lost. Ciliary neuralgia, usually bilateral, of the severest type, and cephalalgia, usually semicranial, are present.

Aside from the increase of tension which may be present secondarily in kerato-iritis and iridocyclitis, acute glaucoma is very rare in childhood. Etiologically, there is a predisposition, shown by hypermetropic structure of the eye, but aside from this no other etiologic factor can be elicited except insufficiency of cardiac function and decrease of arterial pressure.

The prodromal signs are attacks of cloudy vision, rainbow phenomenon upon looking into a candle or lamp light, intermittent feeling of tension in the eye or dull pain radiating to the frontal and temporal regions, less often to the occiput and nape; and often dental neuralgia. These prodromal signs may be absent, and the attack of glaucoma set in without warning.

As to differential diagnosis, parenchymatous keratitis and iridocyclitis should be thought of first. In iridocyclitis, however, the pupil is contracted and irregular, due to synechiæ. In glaucoma, vascularization (pannus regenerativus) is absent, which in children nearly always accompanies the involution of diffuse corneal infiltrates. In neuralgia of the trigeminus, which is exceptional in children, and especially in ciliary neuralgia, the globe is sensitive to pressure. There is neither elevation of tension nor glaucomatous excavation of the optic nerve (Plate XV, Fig. 1). Glaucomatous excavation is characterized, as compared to gray atrophy, by a kinking of the retinal vessels at the optic disc, stagnation of the retinal veins, pulsation of the retinal arteries, and the demonstration of the parallax displacement at the margins of the excavated optic disc.

As to pseudo-glaucomatous excavations of the optic, compare Chapter XVII, 7, closing remarks.

Treatment.—Compare p. 34. Pathological elevation of pressure, without enlargement of the globe, requires iridectomy.

XII. AFFECTIONS OF THE OPTIC NERVE AND RETINA

As the present description is intended for the general practitioner and the pediatricist, those affections only are mentioned which are of importance from a diagnostic viewpoint in infancy and childhood.

The optic disc has only slight connections with the posterior ciliary vessels, while the anterior and middle layers of the retina are dependent upon the narrow retinal vessels, which are devoid of collateral circulation. It can, therefore, be understood that the optic nerve and retina react relatively easily, and with great sensitiveness to every serious impairment of the constitution, and that, especially in the infectious wasting diseases, all kinds of functional insufficiency are noticed.

Visual disturbances are due largely to toxico-mechanical nutritive disturbances of the brain; as, for example, the flitting scotoma with yellow or red vision in influenza, where objects appear as if illuminated by the glare of a great fire; transitory hemianopia in pertussis and scarlatinal nephritis; uræmic amblyopia which, in part, disappears rapidly, and amaurosis during or after measles, scarlet fever, varicella, faucial diphtheria or dysentery; and the concentric restriction of the field of vision in acute infectious diseases, which may undergo rapid involution with the abatement of the fever and during convalescence. In malaria, contraction and extension of the field of vision coincides with the febrile days and the afebrile intervals. So, in croupous pneumonia, rapid involution of the lowered vision sets in with the day of the critical fall of temperature. Pupillary reaction to light points to a central seat of the visual disturbance.

Ophthalmoscopic examination of children is not always easy. When the pupil is very contracted, it should be dilated by one or two drops of homatropine, especially if disease of the vitreous, macular region, or retinal periphery is suspected. It should be remembered that artificial dilatation up to two years is usually very slight. Any intense illumination is to be avoided, in order to have the pupil as large as possible, and to obtain a large, uniformly bright picture that is true to nature. The less infants are dazzled by the ophthalmoscope, the less trouble they give. It is, therefore, well to use a weaker mirror, which will at the same time reproduce more faithfully the natural color of the ocular fundus. The normal ophthalmological picture of the ocular fundus (Plates XI and XII, Figs. 1 and 2) is known in a general way to the practitioner. He also knows that in normal eyes the retinal veins at the optic disc often pulsate, while pulsation of the retinal arteries is pathologic.

In the more or less reddish-white or reddish-yellow color of the nerve-head, an important part is played by the degree of transparency

of the nerve-fibres, the glia tissue, and the cupping, which is found in nearly all normal papillæ, at the entrance and exit of the vessels. The extent of this physiologic cupping presents the greatest varieties. Sometimes it is large enough to approach the lateral margin of the disc. Its walls, however, are not steep, in contradistinction to glaucomatous excavations. In rare cases there is only an uncupped, whitish, closely demarcated, roundish spot at the vascular porus opticus of the disc. The number of capillaries in the tissue of the nerve-head, too, has a bearing upon its color.

Glaucomatous halo, or greenish ring around the optic disc, distortions at the disc due to myopia, myopic conus, circumpapillary atrophy of the chorioid and posterior sclerectasia, must not be confused with the so-called scleral ring (Plate XII, Fig. 2). This light white connective-tissue ring, which completely encircles the disc, is often more strongly marked at the temporal than the nasal margin. There is usually brown or black pigmentation, more pronounced laterally. This pigmented ring (Plate XII, Fig. 2) is narrow in some cases, wider in others, and is caused by a condensation of the pigmented epithelial layer at the margin of the optic nerve. Circumpapillary coloboma of the chorioid and downward conus have already been mentioned on p. 57. There are also polymorphous, diffuse, light yellow reflexes in the infantile eye, distinctly concentric toward the margin of the optic disc, especially near its nasal sector and along the vessels. These reflexes are occasioned by unevenness of the anterior border layer of the retina, and change their appearance and position with rotation of the mirror.

Plate XI illustrates how the pigment content of the chorioid and the pigment epithelium of the retina influence the ophthalmoscopic picture of the ocular fundus. The principal types are: "Albinotic" fundus, poor in pigment; "tabulated" fundus, with chorioid strongly pigmented, pigment epithelium weakly pigmented, causing the very strongly pigmented intervascular spaces to protrude like islets; finely granulated fundus, with dense pigmentation of the pigment epithelium.

It is generally advisable to consult an ophthalmologist regarding all affections of the internal eye, and especially in regard to the optic nerve and retina. The ophthalmoscopic picture of the nerve-head and its vicinity is so variegated that even an expert often requires repeated examinations to determine whether the ophthalmological findings are pathologically important. This is especially true for the diagnosis of an incipient peripheral neuritis of the optic nerve, the initial stage of a choked disc, and the vascular changes of the nerve-head and the retina, the calibre and course of whose vessels show a large number of varieties.

Also compare remarks on pp. 35 and 58.

Aside from a careful history and a thorough general examination,

exact and repeated tests of the central vision (compare p. 286), the color sense, and the field of vision are required. Hirschberger's ocular perimetry with blue and red marks is, as Schloesser emphasizes, to be commended, as, "according to experience, many details will be detected in color examination which cannot be seen in examination with white." Children concentrate better and more quietly on colors, and the examined eye is kept open better, even when the other eye is free. This refers particularly to the determination of central scotoma which, owing to inferior central fixation, can only be indistinctly demarcated or entirely surveyed.

The child holds before the unexamined eye a piece of colored glass, preferably triangular, whose edges are rounded off. The colors used should be complementary yellow in examinations with the blue mark and complementary green with the red mark, so that the marks become colorless for the unexamined eye. In the same way a yellow mark could be eliminated by a blue glass and a green mark by a red one. All these directions, however, should be left to the judgment of the ophthalmologist.

1. HEREDITARY RETROBULBAR OPTIC NEURITIS

The transmission of hereditary retrobulbar optic neuritis is usually indirect, although occasionally collateral, and sometimes direct. It is very apt to be transmitted by females to the male members of the family. It is always bilateral and often accompanied by nervous symptoms, such as vertigo, palpitation of the heart, persistent headache, migraine and sometimes epileptic paroxysms. It never appears during the first ten years of life. Without ophthalmoscopic findings, it suddenly sets in with flashes of light and rapidly increasing cloudiness. This is soon followed by absolute central or a paracentral ring scotoma. Neuritis can but rarely be demonstrated by the ophthalmoscope. There is nyctalopia. Central vision is considerably reduced, while the optic disc gradually becomes pale. A rare phase of the disease is increasing constriction of the field of vision, which usually leads to total blindness. Cure or complete recovery are exceptions.

The *treatment* consists in inunctions of potassium iodide in increasing doses at intervals of from three to six months; brucin injections in the intervals, 1 to 2 mg. per day, spermin (Poehl) and the direct current through an eye cup. From a prophylactic point of view the use of tobacco should be discontinued in later life, on account of the vasoconstrictor effect of nicotine.

2. PIGMENT DEGENERATION OF THE RETINA

(SO-CALLED RETINITIS PIGMENTOSA. PLATE XVI, FIG. 1.)

This congenital degeneration of the retina has already been mentioned on p. 46. It is associated with hemeralopia and ring scotoma and later with high degrees of concentric restriction of the visual field,

especially for blue. Usually it is not distinctly present before the tenth year. From then on there is a gradual limitation of the visual field and failure of retinal function. An important fact in making a diagnosis is that the optic atrophy, progressing with increased thinning of the retinal vessels, is not seen with the ophthalmoscope as a so-called "papilla alba," but as a strikingly yellow decoloration of the optic disc. Syphilitic chorioretinitis, which resembles it, is very often associated with vitreous opacities, while "typical" pigment degeneration, in which the fusiform black spots resemble bone corpuscles, preponderate at first in the retinal periphery and approach the retinal centre only as the disease progresses: nor does it, as a rule, exhibit such a decoloration, or atrophic white spots in the chorioid.

Congenital hemeralopia, due to defective development of the retinal pigment, is probably a prodromal sign of this disease. The pigmentation of the retina, notably in the peripheral equatorial region, is very slight at times ("retinitis pigmentosa without pigmentation"). In other cases the atrophic retina contains only numerous, minute white specks (retinitis punctata albens).

While central vision is at first still relatively good, so that the smallest print can be read, the contour of objects can no longer be readily recognized, as the destruction of the retinal periphery has restricted the visual field. The patient's vision is so constricted that he seems to be looking through a tube. Night-blindness is present from the first, and in very much reduced vision there is nystagmus. Later, posterior polar cataract may develop.

Treatment.—It may be possible at times to delay the loss of sight by constant protection of the eyes from bright light by wearing smoke-gray spectacles and avoiding ocular exertion. Moderate confinement in darkened rooms and strychnine injections in the temples have also been recommended. By treating a forty-three-year-old patient by subconjunctival saline injections alternately into each eye, gradually rising from 2 to 5 per cent., we were able to improve central vision from 5/35 right and 5/25 left to 5/20 in both eyes. The constriction of the field of vision, however (10° in both eyes), remained unchanged.

3. SIMPLE ATROPHY OF THE OPTIC NERVE (PLATE XIV, FIG. 1)

Simple atrophy of the optic nerve may be due to different causes. The relatively most frequent form is gray atrophy occurring in tabes, which progresses steadily. It is very rarely found in children. It begins almost simultaneously in both eyes, with a reddish-gray discoloration and a delicate opacity of the disc. The disc later becomes a pale gray and the nerve quite white; the retinal vessels are moderately contracted.

The resulting visual disturbance has often commenced before a distinct change of the optic disc is visible with the ophthalmoscope. The patient first complains of cloudy vision or more or less troublesome "symptoms of dazzling." The decrease of central vision, too, becomes distinct only later, but early there is red-green blindness. With concentric or irregular, often zigzag constriction of the field of vision, visual power decreases, often unevenly in the two eyes. In the course of one or several years, or even sooner, either gradually or in paroxysms, total blindness ensues. Occasionally some remnant of sight remains in one eye. At first spinal symptoms may be entirely absent.

Elschnig observed ocular crises in this affection, consisting in boring, piercing, drawing pains in the globe and deep orbital structures which, lasting for hours, returned with varying intensity at different parts of the day, and often at intervals of several weeks. They are distinguished from ciliary neuralgia by the absence of contracted pupils and lachrymation at the climax of the paroxysm.

The *diagnosis* is not always easy. Central scotoma also occurs in tabes, with normal conditions of the periphery of the visual field. In incipient tabes, due to chronic empyema of the ethmoid cells and the sphenoid cavity, Elschnig observed retrobulbar atrophy of the optic nerve in spite of energetic antisyphilitic and local treatment, lasting for months, gradually grew worse.

The same author, and others, observed neuritic atrophy with gummatous meningitis, also with simultaneous osseous gumma of the cranium and with simultaneous peripheral chorioiditis and, finally, pallor of both discs due to intracranial affection without evidence of tabes. In doubtful cases it is therefore important to examine the pupils, especially as the Argyll-Robertson pupil is pronounced in the initial stage of tabes, and usually a prodromal sign before the tabetic pallor of the optic disc is evident. The Wassermann reaction, too, often clears up the case.

In tabes, at the onset of visual disturbance, the optic disc assumes a whitish pallor. In descending atrophy, this discoloration due to constricting intracranial and orbital processes (congenital hydrocephalus, etc.) is recognizable only after weeks or months, despite considerable visual disturbance.

In pronounced atrophy the optic disc is porcelain-white and very sharply demarcated—"papilla alba."

However, the ophthalmoscopic picture alone never justifies a prognosis of decline of the visual power. For example, after great loss of blood, I have repeatedly found white discs in both eyes, without particular impairment of the central acuity or the visual field.

First of all, the *treatment* is causal if possible. In pronounced simple tabetic atrophy, energetic mercury treatment accelerates the

decline of visual power. Provided the Wassermann test is positive, the smallest doses of mercury, carefully applied, are less dangerous. A strengthening diet, the alternate use of lecithin in an easily assimilable form, and *syrup. colæ compos.* (Hell), mild hydropathic measures and massage for the promotion of metabolism are to be considered.

Should salvarsan be used, it should be remembered that Spiethoff noticed, about fifty hours after the injection of 0.6 Gm., loss of visual power lasting several minutes, with negative ophthalmoscopic findings, and transitory fluttering scotoma on the day of injection, especially when the patients rose for the first time.

4. VISUAL DISTURBANCES DUE TO LOSS OF BLOOD

Amblyopia is very rare in childhood. It is usually bilateral, and due to gastric, intestinal, or sometimes nasal, hemorrhages. If the amblyopia is slight, it may disappear spontaneously with prompt restoration of the field of vision, which may have been reduced to 3-5. As a rule, the prognosis in high degrees of amblyopia and amaurosis is unfavorable. There is a primary fatty degeneration of the optic nerve and retina, caused by prolonged anæmia of the branches of the ophthalmic artery. I have also observed a permanently and intensely white optic disc in cases running a more favorable course.

Treatment.—As the visual disturbance does not develop until two or three days later, a low position of the head relative to the rest of the body, and bandaging of the lower and upper extremities to reduce the total circulation, may be useful as a preventive. Inhalation of amyl nitrate (1-3-5 drops) and injections of strychnine nitr. (0.001, increasing to 0.003) into the temples have also been tried.

I have observed a remarkable return of vision, outside of the hospital service, in a boy of ten. There had been considerable loss of blood from an injury to the skull which had taken place fifteen or sixteen hours previously, with complete loss of vision in both eyes. Ophthalmoscopic examination being rather difficult, owing to the greatly contracted pupils, I instilled atropine. There was considerable ischæmia of the retinal vessels, especially of the arteries, and a pale, slightly blurred disc. About an hour after the instillation, the boy surprised me with the statement that there was a "shine" before his eyes. I remembered that Schneller had observed dilatation of the chorioidal vessels after instillation of atropine, and I ordered the instillation to be repeated twice a day for a few days. The vision increased from day to day. Examination two weeks later showed improvement of the central vision to 6/24, which rose in the following month to 6/10. The ophthalmoscopic examination showed rather pale discs, and considerable improvement in the size of the retinal vessels. Was this accident? It is not impossible that the

atropine had found its way to the deeper parts, and relieved the spasmic constriction of the retinal vessels. This dilatation reëstablished and activated the impaired circulation in the retina and chorioid.

5. PERIPHERAL NEURITIS OF THE OPTIC NERVE (PLATE XIV, FIG. 2)

This is characterized by opacity, redness and swelling of the optic disc, whose borders are blurred. Etiologically, the following affections may have preceded the optic neuritis: inflammatory intracranial affections, very often congenital syphilitic and tubercular affections of the pia of the brain and spinal cord, orbital abscess, orbital thrombophlebitis, and diseases of the accessory sinuses. In causing primary neuritis of the optic nerve, syphilis and tuberculosis have first place, while grave and febrile general affections much less often play a causal rôle. These forms of neuritis may also be confined to the papillomacular bundle (neuritis fasciculi papillomacularis). In retrobulbar affections at the entrance of the central vessels in the optic nerve, they may manifest themselves perimetrically in the form of paracentral scotomata.

The sequelæ of these transverse and focal affections of the optic nerve are cloudy vision, sensation of darkness, great pain in the frontal and temporal regions, especially at night, disturbance of the central acuity and visual field, demonstrable by functional tests, impairment of the central color sense and color borders. Their development may be rapid or slow. They vary exceedingly and often correspond very little with the equally variable ophthalmoscopic findings, in which, at first, redness and swelling of the optic nerve head and opacity of the retina surrounding the nerve are the most material points. As the process recedes, these manifestations gradually disappear, and dirty reddish-gray or whitish discoloration develops, involving the optic nerve head either wholly or in part (Plate XIV, Fig. 2).

The transition from a blindness to improvement or even to complete restoration of both vision and the color sense is subject to many variations. Disproportionate impairment of the pupillary reaction to light which is not reduced until vision is considerably damaged with a pupil dilated and rigid as in amaurosis suggests cerebrospinal complication from the same etiological factor.

Differential Diagnosis.—Partial or total, finely radial or delicately striated cloudiness of the optic disc is probably often congenital and not very rare. It is due to excessive development of the connective tissue of the layer of nerve-fibres. Other signs of optic neuritis and visual disturbances are absent. Occasionally the cloud is so dense in various areas surrounding the disc that one or the other of the marginal vessels are obscured. In distinction to simple atrophy of the optic nerve, the edges of a disc with secondary atrophy are blurred for a long time.

The retinal arteries are not constricted, as in choked disc, but often even distended. The white spots often present in the retinal centre in neuritis of the optic nerve are not as regularly arranged as the whitish splashes of albuminuritic retinitis (Plate XV, Fig. 2) which surround the macula lutea in a stellate or radial form.

Central scotoma as a sign of neuritis of the papillomacular bundle may also be due to chorioretinitis of the macular region. In that case there is also hemeralopia. Compare remarks on p. 36.

The following peculiarities are noteworthy: Otogenous optic neuritis is, under certain circumstances, the only sign of caries of the petrous bone or of intracranial complications of an otogenous origin (cerebral abscess, purulent thrombosis of the cerebral sinus, etc.).

In meningitic neuritis, the opacity and redness of the optic disc vary with the acuity or chronicity of the meningitis. The other signs in and around the optic disc are just as variable: tortuous and dilated veins, hemorrhages and vascular changes, which with the ophthalmoscope appear as fine whitish striæ accompanying the vessels on both sides.

In syphilitic neuritis of the optic nerve, small radial hemorrhages at the optic disc, and white plaques in the retina, have been observed as signs of perivesiculitis of the smaller vessels.

In tubercular optic neuritis, tubercles have been found at the margin and in the tissue of the disc.

In determining the etiological factor, distinct basilar meningitis and other tuberculous affections are to be considered. The simultaneous presence of chorioidal tubercles is an ominous sign.

Optic neuritis due to affections of the nasal accessory sinuses of (so-called rhino-osteogenous neuritis of the optic nerve) is principally observed in suppuration of the posterior ethmoid cells or of the sphenoid cavity. These air spaces are separated from the optic nerve by an extremely thin osseous wall. Indeed, in certain physiological defects they are almost in immediate contact with it. The disease is acute or chronic, unilateral or bilateral retrobulbar neuritis, attended with papillitis and decoloration of the disc, with central and paracentral scotoma, restriction of the visual field and a reduction of the vision even to total blindness. Since the disc is a process of the dura, deep orbital periostitis, of even a mild degree, or a small quantity of pus between the bone and periosteum occurring in the course of accessory sinus disease, may cause collateral œdema or optic neuritis. Diseases of the chiasma may supervene, leading swiftly to loss of vision. Unilateral affection of the accessory sinuses may produce bilateral diseases of the optic nerve. The sinus disease may require resection of the middle turbinate, endonasal incision and evacuation of the anterior and posterior ethmoid cells, or radical operation on the accessory sinuses. The prognosis is good in

proportion as the process is acute, and the perforation and escape of pus into orbit small. Yet the termination is not always favorable. For example, Beyer observed sudden blindness after affections of the ethmoid bone: "There were polyps in the middle nasal duct without suppuration." But in spite of removal of the polyps, after a short period of improvement, there was an acute exacerbation, with distinct atrophy of the optic nerve, central scotoma and peripheral restriction of vision. The checking of alveolar periostitis of the posterior maxilla due to an affected tooth is usually just as important. Königshöfer reported a case in which neuritis of the optic nerve improved rapidly after removal of adenoids.

If the globe is considerably dislocated, following an affection of the accessory sinuses, there may be a restriction of the field of vision and reduction of the central acuity which will go on to total blindness. These manifestations are due entirely to stretching of the optic nerve. Pressure upon the equator and the lower half of the globe, by changes of refraction in hypermetropia, or by irregular corneal astigmatism, may impair the visual power. If stretching of the optic nerve has not continued too long and has not been too marked, and if there is no affection of the vascular apparatus or an infectious neuritis, and no pressure from the swelling in accessory sinuses upon the optic nerve, the visual disturbance may gradually undergo complete involution.

Optic neuritis in acute myelitis usually ends with atrophy. Restitution of vision is the exception. Visual improvement is often marred by permanent disturbance of the color sense, and, with even relatively slight findings, the visual disturbance which may precede the eruption of the primary disease is at once uncommonly severe.

In epidemic cerebrospinal meningitis, amblyopia occurs with negative ophthalmoscopic findings at first. Atrophy of the optic nerve becomes apparent only at a later stage.

In visual disturbances from diseased upper teeth, neuritis of the optic nerve is caused by acute alveolar periostitis, which spreads either along the nerve sheaths to the orbit and optic foramen, or through the abundantly developed vascular net of the alveolar processes of the superior maxilla to the venous system of the orbit. The alveolar plexus in rare instances anastomoses with the pterygoid plexus and the ophthalmic vein. The resulting disturbance of circulation is shown not only in neuralgia of the trigeminus, but also in collateral œdema of the maxillary sinus, in slight œdema of the face or lids of the affected side and of the optic nerve. Invasion of the latter is likely to take the form of an acute neuritis of the papillomacular bundle. This is functionally apparent as a central scotoma for white and colored marks, with reduction of the central visual acuity.

Affections of the brain and spinal cord of all kinds are the connecting links between a number of visual disturbances and other ocular changes which may appear sooner or later, sometimes after many years. Meningitis in particular is often regarded as the cause of optic neuritis after measles, scarlet fever, typhoid, and pertussis.

Unilateral or bilateral visual disturbances in erysipelas and retrobulbar orbital inflammations are occasioned by compression of the vessels or thrombosis of the central veins, by inflammation of the optic nerve, and retinal hemorrhages. It often leads to total blindness.

As to neuritis of the optic nerve in sympathetic ophthalmia, see chapter on "Injuries."

Hemorrhages in the optic nerve itself, sometimes leading to effusions of blood into the vitreous, may occur in pertussis, influenza, variola, and in the scorbutic stage of leprosy.

A sudden visual disturbance as a sequel to acute retrobulbar neuritis in influenza, measles, scarlet fever, pertussis, or typhoid fever may rapidly end in total blindness. There are either dilated pupils without reaction to light, slight congestion of the retinal vessels, or a picture corresponding to embolism of the central artery, and in some cases pronounced papillitis. The children complain of violent headache or dull pains in the orbit, which increase with ocular movements or pressure upon the globe. Vision may improve sooner or later, with abatement of the pains and restitution of pupillary reaction. Central scotoma is noteworthy in the stage of involution. The ophthalmoscopic picture of atrophy of the optic nerve develops later, perhaps with pigmentation of the optic disc. The process is often the result of a compression of the optic nerve by a retrobulbar hemorrhage.

Treatment.—The primary affection must be treated. The eyes are to be protected from bright light, and should not be subjected to undue visual efforts.

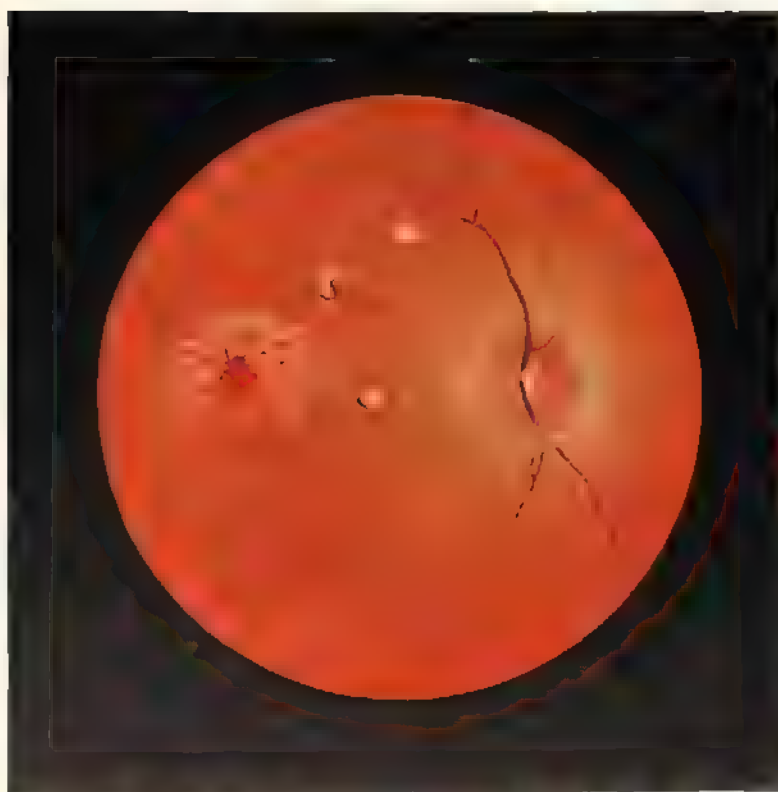
Local: In acute neuritis of the optic nerve, the intermittent application of the Bier-Klapp aspirating globe, as modified by Peters (Fig. 37), is recommended, provided there are no signs of anæmia. It is particularly efficacious if, as in odontogenous neuritis of the optic nerve, there are evidences of stasis in the retrobulbar segment of the optic nerve.

The glass cup is preferably applied in the recumbent position, the larger one to the temple and the smaller one behind the mastoid process. To prevent its falling off, an adhesive salve is applied to the edge and to the corresponding part of the skin. The resulting visible changes of the skin vary considerably. It is drawn up more or less rapidly and, after the glass cup is removed, the skin has either a uniform darker hue, or is infiltrated with numerous, small, petechial hemorrhages. If long continued, a circumscribed blister of the skin, as if from a burn, may be formed by

PLATE XV



Glaucomatous excavation



Albuminuric neuroretinitis

accumulation of serous fluid which upon the bursting of the vesicle more or less empties itself. The procedure should not be repeated until the place of rupture has healed, which will be the case in two or three days, a mild ointment having been applied. It is desirable, therefore, to apply the aspirator three times for five minutes at intervals of five minutes, alternating with ear and temple cupping and increasing by a few minutes with each application the duration as well as the interval. This prevents the parts becoming so accustomed to the effect as to minimize results.

Aspiration, like dry cupping, causes a vascular contraction with subsequent hyperæmia. In order to keep this within adequate bounds and to prevent injurious results, such as increased excitement and headache, the patient keeps his eyes closed for an hour after the application. The treatment is best carried out twice a week between five and six o'clock in the afternoon. A weak smoky-gray pair of spectacles is worn for the following twenty-four hours, and the diet should be light.

It is equally important to eliminate toxic products by mild hydropathy and diaphoresis, as explained on pp. 234, 235. Inunction treatment and the iodides are useful, even if there are no indications of syphilis. I always prescribed them as a precautionary measure, with an addition of cœrumentum. (ung. hg. ciner. with cœrument. $\bar{a}\bar{a}$ in small doses twice to three times daily, $\frac{1}{2}$ -1 Gm. of the mixture at a time). For the advanced atrophic stage, however, this treatment is not always appropriate. A better treatment consists in injections of equal parts of brucin and fibrolysin into the temporal skin in gradually increasing doses. The first remedy increases the excitability of the retinal elements and the second prevents constriction of the optic nerve. Injections of saline or hetol solutions into the capsule of Tenon are also appropriate. Silex recommends the direct current, applied through an eye cup. Quinine should be used cautiously, as it has occasioned vascular spasms which have often led to considerable thinning of the retinal vessels, paling of the optic disc and sudden blindness. The latter can be improved, but a certain degree of asthenopia and especially a restriction of the visual field will often persist.

The treatment in amblyopia and amaurosis without ophthalmoscopic findings is on the same line.

In pertussis, relief of the coughing paroxysms by bromoform, pertussin, etc., is important in order to prevent intra-ocular hemorrhages.

VOL. VII—18

FIG. 37.



Suction apparatus (after Bier-Klapp) in operation.

6. TOXIC AMBLYOPIA

Toxic amblyopia, caused by the extract of male fern, is of practical importance in children. This remedy is often given for the removal of tænia or hookworms. Its use is not without danger in nervous, irritable and weakly children, especially when its administration is immediately preceded by the usual routine treatment of debilitating diseases like influenza. The combination of the extract with castor oil is especially dangerous, as oils enhance the toxicity, and such medication may be followed by sudden and serious injury to the optic nerve.

In a case which I observed, the use of a patent medicine, which contained 10 parts of the extract of male fern and 90 parts castor oil, caused headache and vomiting, followed by sleep lasting about 36 hours, from which the patient awoke totally blind. Examination showed dilated pupils without reaction, hyperæmia of the optic disc and constriction of the retinal arteries. The light of the largest lamp was no longer recognized. There was a pronounced albuminuria for a few days. Treatment was unsuccessful and atrophy of the optic nerve developed.

7. SYPHILIS

In addition to what was said concerning syphilis it must be added that neuritis of the optic nerve may be the only sign of congenital syphilis. As the diagnosis is often difficult in the early stage, prompt ophthalmoscopic examination by a specialist is advisable in every case of congenital syphilis.

According to Japha, changes of the ocular fundus not easily recognized by inexperienced observers occur in about 66 per cent. of nurslings with congenital syphilis. They may be the only sign of affection besides the Wassermann reaction. In etiologically doubtful nerve disturbances in infancy, due to mild meningitic irritation (spasms, unexplained restlessness and crying), they may determine the diagnosis.

According to Hirschberg, the optic disc and the surrounding retina is at first opaque. Soon after, there are light specks over the entire fundus which in the course of time increase in number and size and acquire a fine punctiform pigment. At an early stage the retinal centre becomes gray, but later it may again become pale. The periphery looks as if paved with light, checkered or dark foci.

In Japha's observations these changes are of slight importance, as compared to the neuritis. He examined every case of congenital syphilis for ocular changes, so far as possible, and arrested the disease at the onset. Hirschberg observed grave changes, such as vitreous opacity, connective-tissue formation in the vitreous, detachment of the retina, degeneration of the central retina, and connective-tissue contraction of the optic nerve. Of all these Japha saw only a few cases of vitreous opacity and

twice optic atrophy with total or nearly total blindness. The affection of the fundus was usually but not always bilateral. Attentive mothers have occasionally observed a peculiar rolling of the eyes, blinking or squinting. Other symptoms, according to Hirschberg, are an oblique position of the head, and nystagmus. Many infants protect the eyes with their hands on account of photophobia. When the disturbance is serious, they no longer reach out for objects held before them, and children who can walk bump against everything when the light is bad. Generally speaking, however, all visual disturbances that occur are of a grave nature.

The prognosis is quite favorable, provided treatment is timely. Many cases improve rapidly, especially if neuritis only is present. True, the optic nerve does not always become quite normal again,—in fact, it may turn white,—but the visual acuity actually present shows that the ophthalmoscopic findings are no criterion for visual acuity.

Hirschberg uses inunctions, repeated if possible at intervals through the first two years of life, but never more than one hundred applications in all. Japha prefers internal medication in two or three treatments of fifty days each, the dose consisting of 0.005 to 0.01 proto-iodide of mercury, twice daily. Rarely is it necessary to continue treatment longer or to resort to injections. The affection may sometimes take a favorable course even without treatment, but the possibility of grave changes and considerable impairment of the visual acuity is increased.

As to the use of salvarsan, compare remarks on p. 268.

8. ALBUMINURIC RETINITIS (PLATE XV, FIG. 2)

Visual disturbances due to fundus changes of nephritic origin may develop either gradually or with inordinate rapidity. When the process is gradual, patients complain of transitory disturbance of vision, or of flickerings of light; although even in extensive retinal changes these disturbances may be so slight that they appear as a cloudiness, especially noticeable upon reading small print. On the contrary, quite a reverse picture may be presented. The visual power may also change without producing any changes in the ophthalmoscopic appearance. Central vision is most seriously impaired in hemorrhages of the macula lutea or the vitreous, in neuroretinitis, and in involvement of the optic nerve, which later becomes atrophic.

Leaving out of consideration amaurosis, either uræmic or due to great loss of blood or simultaneous infectious processes, total blindness occurs only with thrombosis of the cerebral arteries and hemorrhages of the optic nerve, such as may be observed in retinal detachment, in interstitial nephritis of measles and in fatal cases as a premonitor of death.

Blindness may also be due to intercurrent gastric or intestinal hemorrhages (see p. 268).

Unilateral occurrence of the disease is rare, but often both eyes are involved in different degrees. The ophthalmoscopic pictures are rarely as distinct as in typical albuminuric neuroretinitis.

The true degenerative retinal changes are the most frequent, and vary in number, distribution and size. They appear as gray-white, and sometimes silver-white dots, specks, lines or spots, having either a dull, lustrous or shining appearance. They preponderate about the optic disc and around the macula lutea, where they often assume a stellate or radial arrangement. At the onset these changes are often so minute that the areas have a finely granular appearance. Later, the larger spots fuse, forming white foci and surrounding the disc in the form of a ring or rampart which more or less hides the retinal vessels, whereupon the macular region has a whitish appearance and is covered with a dark brownish veil. On, or in the vicinity of, the spots, which are due partly to oedematous swelling or necrosis of the nerve-fibre and ganglionic layers, partly to fatty or hyaline degeneration of the external reticular and the two granular layers, there are found linear hemorrhages, which more or less hide the larger retinal vessels. When the hemorrhages are large, they often have a flaring appearance. Circular or irregular hemorrhages occur also in the deeper layers of the retina toward the periphery. Much more rarely hemorrhages are found in the shape of pools of blood between retina and vitreous. These occasionally perforate into the vitreous and may then interfere with a detailed fundus examination. Hemorrhages are occasionally so large and numerous that the white spots recede or are confined to the region of the fovea. Either all or part of the remaining retina may be much smaller, and of an opaque gray color.

The entrance of the optic nerve is often hyperæmic or shows a reddish-gray discoloration, and the disc appears cloudy, causing the adjacent retina to become less transparent.

Optic neuritis may be the only change, or may be so pronounced that it presents the picture of a choked disc. There are also slight signs of retinal degeneration, the most numerous of which are in the vicinity of the macula lutea. Optic neuritis in one eye and albuminuric retinitis in the other is not extremely rare.

The retinal veins are more congested and often tortuous, and the arteries narrower than normal. In very advanced nephritic processes, owing to oedematous swelling of the retina, the non-constricted arteries seem to have completely disappeared in certain areas, while the congested and tortuous veins, resembling linear hemorrhages owing to the absence of the central reflex striæ, are less obscured.

In the majority of fatal cases all these changes either persist or

increase until death. In less grave cases of acute nephritis amenable to treatment and which develop from a chronic affection of the kidney, the visual disturbances are often cured.

The deeper white spots, the splashes, and spots around the macula lutea are the most obstinate. The hemorrhages, which may frequently reoccur, either disappear by fatty degeneration without leaving a trace, or they leave a very slight pigmentation of the corresponding retinal region. Optic neuritis may lead to total pallor of the disc, though with relative restoration and persistence of the visual power. It may be difficult to make a diagnosis of a nephritic affection of the retina and optic nerve which has terminated a long time ago. The best guides are the thinned areas and whitish sheaths of the retinal arteries, retinal cords and atrophic spots of the pigment layer and optic disc.

The changes in the chorioid (which is always involved) are usually inaccessible to exact ophthalmoscopic examination, owing to the opacity of the optic nerve and retina. Visible circumscribed or disseminated chorioiditis appears in the shape of delicate, yellowish-pink or whitish-yellow spots of various sizes and shapes, fringed with pigment. This is especially true in the equatorial sections of the fundus. In pronounced cases they can still be recognized later on as pigmented spots and scars. Hemorrhages may occur in the chorioid, which lead to subsequent atrophy and pigment changes.

Detachment of the retina, unless complete, causes a partial defect in the visual field. It usually follows chorioidal disease, and, while it rarely appears as an early symptom of the primary disease, it is more frequently a late manifestation, setting in shortly before death. It may then be associated with vitreous hemorrhage.

The detached retina is usually very opaque, with vascular protrusions, and folded but slightly if at all. It floats only if the hemorrhage has perforated into the vitreous, or there is liquefaction and subsequent shrinking of the vitreous.

Considering that nephritic trophic disturbances of the fundus which may be seen by the ophthalmoscope are often confined to the macular region, and may not even be recognizable except in the upright image, dilatation of the pupil for diagnostic purposes is to be urged, as an early recognition of these changes is important.

Differential Diagnosis.—The changes in the optic nerve and retina, as have been described, also occur just before or coincident with tuberculous, acute or subacute leptomeningitis and increased intracranial pressure due to tumor. The fact that in brain tumor swelling of the optic disc is quite marked is of no vital importance, any more than the fact that the stellate figures of the macula in the papillitis, with meningitis, do not usually occur until the evolution of inflammatory process.

According to Hirschberg, a more decisive diagnostic point is the fact that choked disc, due to a cerebral tumor, occurs before the visual disturbance, and in its initial stage is always accompanied by good vision.

Should choked disc and paralysis of the cerebral nerves be present simultaneously, both may have been caused by tumefaction as well as hemorrhagic pachymeningitis. On the other hand, a little albumin may be found in the urine in the presence of a cerebral tumor, even without nephritis.

A characteristic sign of medullated nerve-fibres is the permanency of the ophthalmoscopic picture. There are neither hemorrhages nor degenerative changes in the optic nerve and retina. Of course, both may occur simultaneously.

In differentiating the glistening white spots in the posterior staphyloma of myopia (which may also show a radial arrangement in the macular region), it should be noted in the latter the sclera appears denuded, and has a sinewy lustre which increases in distinctness in proportion to the pigmentation of the surrounding chorioid. Here and there retinal vessels of an entirely normal appearance as to color, repletion, and walls may pass over these areas. Should there also be fatty degeneration of the chorioid, typical cholesterol needles can frequently be seen with the ophthalmoscope.

Prognosis.—Affections of the ocular fundus always point to grave affections of the vessels and kidneys. The progress is comparatively more favorable if the visual disturbance and retinal changes set in with the primary affection or as an early symptom.

The individual character of the primary disease, the physical condition of the rest of the body, and the degree to which general treatment proves efficacious are factors of importance.

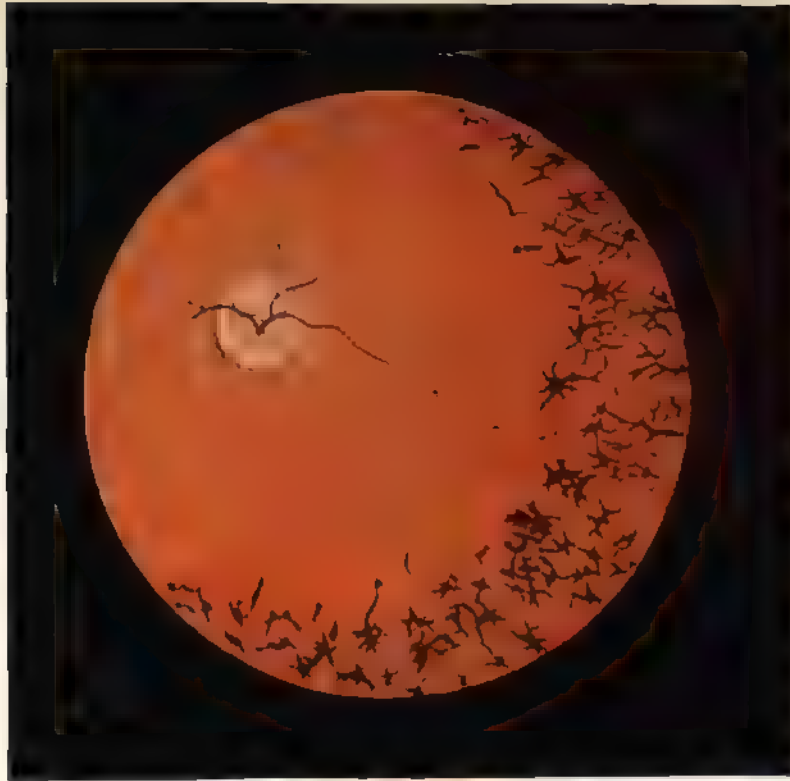
Treatment.—Local treatment is only exceptionally needed and then for the prevention of glaucoma by occasional instillations of physostigmine-pilocarpine (physostigmin. salicyl. 0.025, pilocarpin. mur. 0.05, sublimate, 1 : 5000, 10.0; ad vitr. Nigr. opt. claus. Sig. One drop to be instilled in the evening before retiring).

In detachment of the retina in a favorable case, resorption of the exudate, by diaphoresis, should be considered; but consultation with an experienced ophthalmologist is imperative. As a rule, the first weeks of treatment will show whether a reattachment of the retina may be expected.

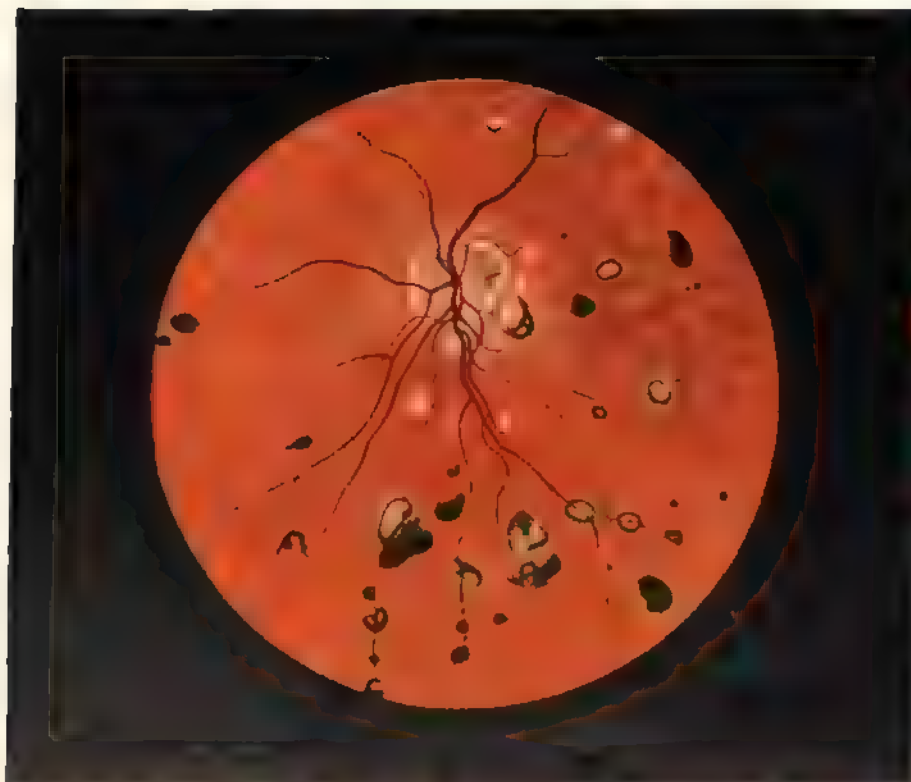
Violent headaches may be relieved by application of an ice-bag to the head.

Albuminuric retinitis should not be confused with "septic retinal changes" due to toxicity or capillary obstruction; these latter are very

PLATE XVI.



Retinitis pigmentosa.



Disseminated choroiditis.

important for the differential diagnosis between typhoid fever, meningitis, miliary tuberculosis and septicæmia, or pyæmia. There are hemorrhages and yellowish-white spots in the disc and retina, notably at the places of venous bifurcation. They appear soon after the onset of the general affection, new spots appearing while the old ones undergo involution. They may disappear without a trace, if the general affection is thoroughly cured. Thus they are of no value in prognosis.

9. CHOKED DISC (PLATE XIII, FIG. 2)

Changes in the optic nerve the result of nephritis should not be mistaken for choked disc. Aside from meningitis choked disc occurs in intracranial new growths, especially in tumors of the cerebellum and the posterior fossa. With the latter tumors choked disc occurs much earlier and more regularly than with new growths in other parts of the skull. This is due chiefly to the increased lymph stasis produced by the tightly drawn tentorium cerebelli.

In cerebral tumors developing after cranial injuries, in addition to choked disc there are other cerebral symptoms, especially traumatic paralysis of the extreme ocular muscles and the auditory nerve.

Visual disturbances occasioned by choked disc may include even sudden blindness. They may, however, be caused also by pressure of a new growth upon the aqueduct of Sylvius, especially in the cerebellum, with subsequent dropsical dilatation and hernia of the floor of the third ventricle, which in turn produces compression of the chiasma and the optic tract.

Choked disc as a sequel of cerebral abscess, internal hydrocephalus, meningeal hemorrhages, and extensive cerebral aneurisms is much rarer. The attendant visual disturbances only come to the notice of the patient in an advanced stage, and in many cases not before the climax of the papillitis has passed and the first signs of degeneration of the optic nerve have become visible.

For the *differential diagnosis* it is important to note that the choked disc in these cases is seldom as markedly developed as in cerebral tumor.

Capillary hyperæmia of the optic nerve, neuritis, neuroretinitis, and choked disc in suppuration of the middle ear occur in otitic abscess of the temporal lobe and cerebellum. Optic neuritis in these cases is usually bilateral and more pronounced on the affected side.

The visual disturbance, although not very considerable, does not disappear at once after evacuation of the abscess; indeed, it may progress further. Amaurosis or atrophy of the optic nerve almost never occurs, unless complicated with pronounced internal hydrophthalmus or, as is the case in cerebellar abscess, with ventricular serous meningitis.

These changes in the optic nerve are much rarer in otogenous ex-

ternal purulent pachymeningitis or sinus thrombosis and in affections of the middle ear and the mastoid without serious intracranial disease. It may be absent even in the presence of a thrombosis of greatest possible extent and extradural suppuration. Postoperative choked disc without retinal nodules is a transitory occurrence in spontaneous cures.

Thrombosis of both cavernous sinuses does not, as such, cause choked disc, not even when a large portion of the orbital veins is involved. Occurrence of choked disc in this connection indicates the presence of a space-restricting intracranial complication which may be operable.

According to Schieck, choked disc may be caused by the increase of cerebrospinal fluid which, under pathologically increased pressure due to intracranial or intra-orbital processes, finds an outlet from the inter-vaginal space in the preformed lymph-sheath of the axial cord along the central vessels, enters into the disc from behind.

Treatment.—The first important step is the reduction of intracranial pressure, since its elevation is in a large measure responsible for papillitis dependent upon intracranial conditions. Potassium iodide and mercury, if there is intracranial gumma or cerebral syphilis, salvarsan, removal of tumor and abscess, are the indicated remedies. Palliative trephining was suggested in Germany first by E. v. Hippel and adopted enthusiastically. If done in time, especially in papillitis of non-syphilitic origin, it may not only prevent impending loss of sight, but prolong and even preserve the life of the patient.

Thus, A. v. Hippel saw a boy of ten years who developed papillitis of the right eye and incipient papillitis of the left eye, accompanied by violent headache, rigidity of the neck and paresis of the left abducent nerve. Localization of the pathological focus was impossible. Trephining effected a complete cure, restoring both discs to normal and vision to 1.0 right, 0.9 left, as against 1.0 right and 0.8 left before the operation.

If acute or chronic otitis media is the starting point of the affection, repeated ophthalmoscopic examination and the services of an otologist are of urgent importance, even if in the beginning of ear disease the optic disc has a normal appearance with a mere pathological redness.

10. CHANGES OF THE OPTIC NERVE IN OXYCEPHALIA

The vast majority of the cases so far observed were males. There was either a premature ossification of the cranial sutures or a thickening of the osseous substance owing to precipitated growth or exaggerated nutritive processes, as in physiological ossification, hyperæmia and osteitis in R. Virchow's sense.

The indirect or-direct subsequent symptom in the shape of optic nerve atrophy was post-papillitic or post-neuritic in forty cases, while primary atrophy appeared but twice. It is not yet clear whether the

process in the optic nerve is the result of a "universal elevation of intracranial pressure," caused perhaps by a coördinated external hydrocephalus accompanying the osseous process, or of a pachymeningitic or an optic meningitic process, in which involvement of the basal course of other cerebral nerves was also observed. Abnormal constriction of the optic foramen with compression of the optic nerve has been suggested. Behr refers choked disc in this connection to peripheral lymph stasis, because the carotid is displaced into the posterior half of the optic canal by the shifting of the bones which takes place in oxycephaly, pushing the optic nerve against the wall. In some cases there was prominence, in others flatness of the orbit, hyperopic refraction and divergence of the globes. In a boy of two and a half years Uhthoff found bilateral choked disc associated with a high degree of *impressiones digitatæ* of the cranium, and shortening of the orbit and frontal position of the superior orbital wall, which was demonstrated by the X-ray. However, pronounced pyrgocephalus also occurs, with or without exophthalmos and with tolerably good central vision, in spite of concentric restriction of the visual field.

In neuroretinitis or in choked disc with general elevation of cerebral pressure, treatment may be by palliative trephining or lumbar puncture.

11. GLIOMA OF THE RETINA

This is exclusively an affection of infancy occurring between the first and third years. It is usually unilateral,—according to Adam in 8.5 and to Wintersteiner in 24 per cent. of the cases. Both sexes are affected about equally. Glioma occurs relatively often in several members of a family, but the part played by heredity is not determined.

The period between the affection of the first and second eye varies between six months and five years. The diagnosis is rarely made until the new growth has advanced to such an extent that its surface in the vitreous is noticed by the child's relatives as a bright reflex coming from the depth of the eye. It can be recognized as retinal glioma with mere lateral illumination. The growth is at first yellowish-white, straw-yellow or reddish-yellow, with roundish or nodulated eminences having newly formed vessels and presenting a picture formerly described as amaurotic cat's eye. It continues to grow forward with progressive detachment of the retina. The vitreous space is gradually filled to such an extent that the lens closely approaches the cornea, and gradually becomes so opaque that the interior of the eye can no longer be inspected.

The second stage soon follows. It is characterized by chronic inflammatory glaucoma and incipient enlargement of the globe. Subsequently the third stage sets in, consisting in perforation of the globe and

extrabulbar extension. The latter will be found either at the posterior pole, in the anterior parts of the globe, or along the optic nerve, or simultaneously in both places.

The *diagnosis*, even in the very beginning, generally presents no difficulties to the experienced ophthalmologist. As to the differential diagnosis, "pseudoglioma" (Plate VIII, Fig. 5), abscess and connective-tissue degeneration of the vitreous, intrabulbar or uveal tabes, cysticercus in the vitreous body, and an affection located at a persistent hyaloid artery, all possible alternatives. In doubtful cases, a blind eye of this description should be removed.

It is easy to determine whether the retinal detachment is due to a different affection, if the detachment is circumscribed and the retina still transparent.

Detachment may also be occasioned by a sudden chorioidal hemorrhage of exudative chorioretinitis (as in nephritis). It may also be due to chorioidal sarcoma and ciliary tubercles, to subretinal cysticercus, contusion of the globe, cord formation in the latter, and tearing or detachment of the retina at the ora serrata. The latter is particularly liable to occur in a longitudinal, ovoid eye with staphyloma posticum.

In exudative chorioiditis, a change in the position of the head may also change the position of the detachment. Transillumination of the globe from the sclera or from the oral cavity should be used when it is doubtful whether the trouble is uveal melanosa or hemorrhagic cyst in the ciliary body.

The behavior of the intra-ocular pressure is less useful for purposes of diagnosis. If the detachment is fresh, tension may be near the maximum as well as the minimum limit of normal. When the so-called exudative-serous retinal detachment, which usually is partial at first, becomes total and leads to iridocyclitis, there is nearly always hypotony.

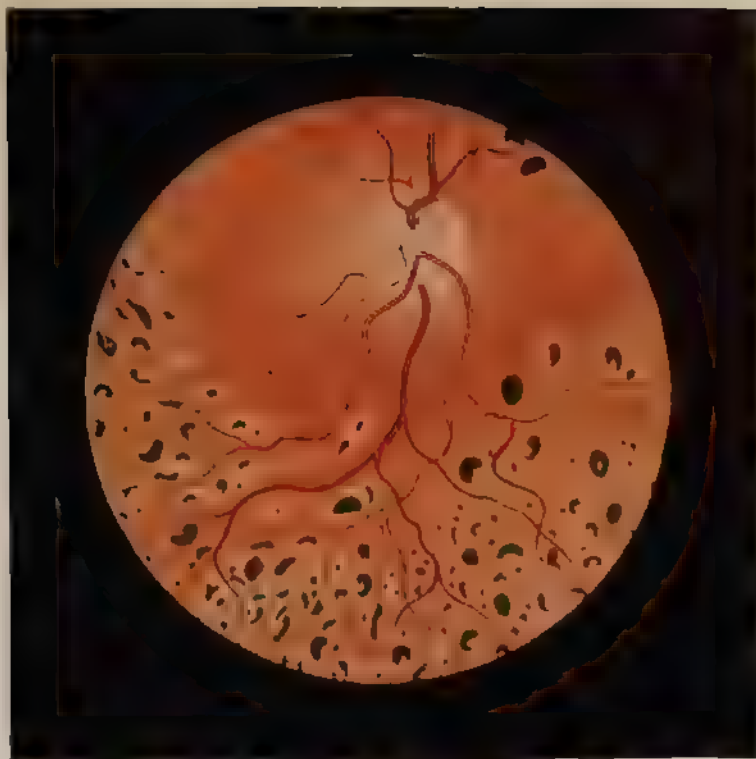
The *prognosis* is favorable, so long as the glioma is restricted to the retina. It becomes less favorable if it spreads to the optic nerve and chorioid. It is unfavorable, even after exenteration of the orbit, if the neoplasm extends beyond the globe, as there will soon be recrudescences which will cause death by cerebral metastases.

According to Hirschberg, the prognosis of the operation is bad, even in the early stages.

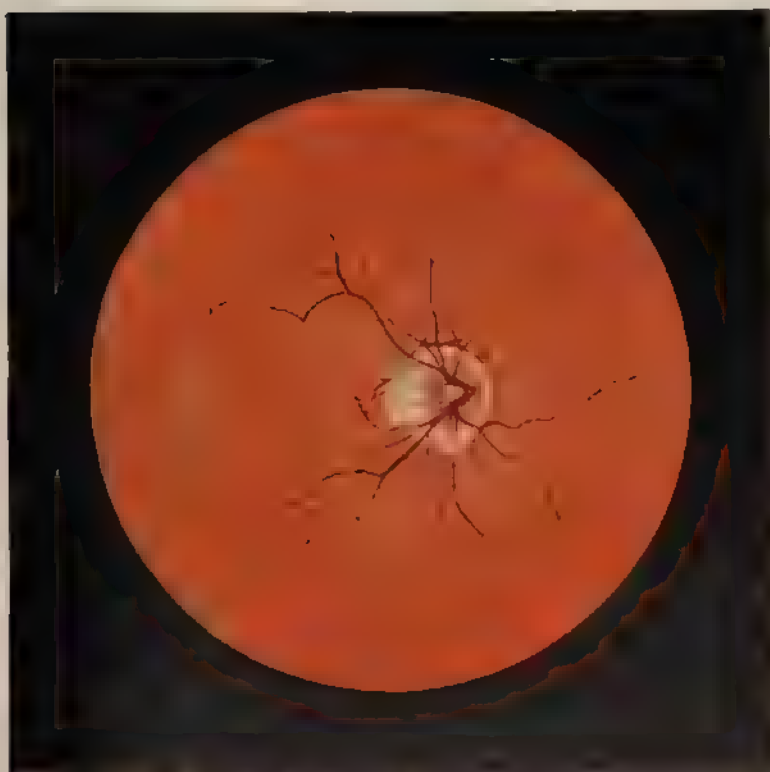
Extension of the tumor to the optic nerve does not seem to be quite so hopeless, since enucleation and evisceration have often led to a cure. The mortality is greatest in the first years of life.

Treatment.—In the first stage, in which the glioma has not yet spread beyond the retinal border, and in the second stage, which is characterized by beginning enlargement of the globe, enucleation is sufficient. In cases in the second stage which have persisted for more

PLATE XVII



Congenital syphilitic chorioretinitis.



Circumpapillary atrophy of the choroid (myopia).

7

than three months, and in the third or extra-ocular stage, exenteration of the orbit is necessary. Of course, in the third stage evisceration is merely a palliative measure except where the extra-ocular extension has been confined to the optic nerve. The latter cases are cured by enucleation of the globe and resection of the nerve.

However, the dividing line between the second and third stages may be uncertain, unless infiltration is visible at the corneoscleral border, with impending perforation at the anterior part of the globe. But in cases which clinically seem to belong to the second stage, the anatomical symptoms point to the third stage and posterior perforation, such as reduction of the gliomatous eye, excavation of the sclera, reduced motility, chemosis and exophthalmos, hypotony with normal bulbar tension in spite of buphthalmic symptoms, choked disc or compression atrophy of the other eye.

The consent of the relatives to evisceration must be obtained before operation, as Adam pointed out, because after enucleation an inspection of the removed globe may disclose extra-ocular extension and render immediate exenteration of the orbit necessary. Enucleation in these cases means resection of the insertion of the four rectus muscles, which is done by drawing the globe through the lid space, and resection of the optic nerve by vertical insertion of the scissors as nearly as possible to the optic foramen.

The decision as to whether operation should be resorted to in bilateral glioma is best left to the parents.

12. OTHER TUMORS OF THE OPTIC NERVE

Aside from glioma, which also occurs as a primary retrobulbar affection, there are other neoplasms of the optic nerve in infancy and childhood, notably fibrosarcoma, myxosarcoma, angiosarcoma, and the infectious granulomata (tubercle, gumma). They often develop first in the orbital part of the optic nerve and lead with early blindness to a usually painless, slow displacement of the globe, either directly forward or slightly downward and outward. The blindness is sometimes accompanied by neuroretinitis or optic atrophy. The motility of the globe in all directions may be undisturbed for a long time, but as it continues to protrude, the lid's movement may be interfered with to such an extent that lagophthalmos and keratitis may develop from desiccation.

Treatment.—Removal of a tumor, either by the anterior route, by detaching the interior rectus muscle (H. Knapp), or by osteoplastic resection of the external orbital wall (Krönlein), at the same time preserving the eyeball, has been successfully carried out. These procedures would be indicated in tuberculosis of the optic nerve only in exceptional

cases. Gummatous infiltration of the optic nerve with considerable involvement of its ocular end produces ophthalmoscopically a cloudy, yellowish-white structure protruding into the adjoining vitreous, with or without hemorrhages. Energetic specific treatment of this condition may effect a striking improvement. In a case of bilateral blindness, with negative ophthalmoscopic findings, but where there was probably an injury to the deep parts of the right orbit or at the cranial base, I prescribed inunctions which resulted in a complete cure of the left eye and considerable improvement of vision in the right eye. In this case there was great pallor of the optic disc and permanent perivascularitic sclerosis of the papillary vessels.

As to injuries of the optic nerve and retina, and their sequelæ, see chapter on "Injuries."

XIII. ANOMALIES OF REFRACTION AND ACCOMMODATION

ANOMALIES of refraction and accommodation produce more or less important visual disturbances for both distant and near points. The normal eye, when accommodation is relaxed, perceives objects "infinitely" distant perfectly. In emmetropia parallel rays of light are brought to a focus exactly upon the retina, nor can the retinal picture be made more distinct by any artificial means, such as concave, convex, or the other lenses.

From the standpoint at differential diagnosis it must be remembered that visual disturbance may also be occasioned by factors other than refractive anomalies, as, for example: opacity of the refracting media; anomalies of color perception. The same holds good for defects of color and light sense or excitability of the light-perceiving apparatus of the retina (retina, neuro-epithelium), the conducting nerve-fibres, and the complex cerebral centres particularly concerned in vision, and for disturbances of the photochemical apparatus (neuro-epithelium of the retina, pigment epithelium).

The degrees of refraction anomalies are determined objectively by skiascopy or ophthalmoscopically with the upright image, and subjectively by determining vision for distant and near points by means of reading tests with spherical and cylindrical lenses. As children have a marked tendency to accommodate, the test, especially for myopia, may have to include an examination with homatropine and the use of stenopaëic discs.

When estimating refraction with the ophthalmoscope by means of the upright image, the examiner who has learned to relax his accommodation completely must bear in mind that if he has an error of refraction, for example myopia, he will need to have his own refractive error corrected in order to see the fovea or temporal border of the optic disc. Should the lens required for a clear view be of more diopters than that needed to correct the examiner's error, the difference between the two lenses corresponds to the degree of the myopia actually present in the patient's eye. If the corresponding glass is 10 D. weaker than his own ametropia, the examined person has 10 D. of the opposite refractive error.

Practical tests for visual acuity are always made at a distance of at least 5 metres in order to approximate parallel rays from the test object. As the correcting glass thus found to be best does not fall upon the nodal point of the eye, it is correspondingly stronger in myopia and weaker in hyperopia than the actual degree of myopia or hyperopia. If, for

cases. Gummatous infiltration of the optic nerve with considerable involvement of its ocular end produces ophthalmoscopically a cloudy yellowish-white structure protruding into the adjoining vitreous, with or without hemorrhages. Energetic specific treatment of this condition may effect a striking improvement. In a case of bilateral blindness with negative ophthalmoscopic findings, but where there was probably an injury to the deep parts of the right orbit or at the cranial base, prescribed inunctions which resulted in a complete cure of the left eye and considerable improvement of vision in the right eye. In this case there was great pallor of the optic disc and permanent perivasculitis and sclerosis of the papillary vessels.

As to injuries of the optic nerve and retina, and their sequelae, see chapter on "Injuries."


instance, a distant point of 22 cm. was found and the correcting concave glass was 2 cm. this side of the nodal point, there is myopia of about 4.5 D., requiring a glass of 5 D. (22 cm. - 2 cm. equals 20 cm.). By increasing the distance from the eye, convex glasses have a stronger and concave glasses a weaker effect. In higher degrees of ametropia it is necessary, therefore, to take the distance of the correcting glass from the eye into consideration.

If spectacles have been worn before, their refractive power is first determined by the direction of the displacement of objects viewed through it when the lens is moved up and down. If the lens is convex, any objects so seen through it (crossbar of a window, or candle) will move in the opposite direction; but when seen through a concave lens it will move in the same direction.

The strength of the lens examined is indicated by whatever spheric convex or concave lens neutralizes or, in other words, converts it into a plane lens, so that there is no displacement upon movement.

The refractive power of the eyes is subjectively determined by the strongest convex and the weakest concave glass with which the best central vision is attained on the test chart. The test must be conducted at a distance not less than 5 metres, and in a room uniformly lighted with natural light.

Central visual acuity (so-called central vision) is tested in diffuse, uniform, and favorable daylight by the ability to distinguish two separate spots, black in white or white in black. According to Aubert, an object can be seen which produces a retinal image of 0.0025 mm. in size.

Ever since Aubert, Snellen and Giron-Teudon, it has been agreed to accept as normal, test objects or types built up of units which subtend a visual angle of 1 minute. The reading test-boards first used upon this principle contained single figures or letters, the lines of which represented a thickness of 1' for the distance to be considered, while the whole test objects appeared under an angle of 5'. But since some of these test objects were more easily recognizable than others, and were therefore guessed at to a certain extent, Snellen substituted hooks with three prongs, , and Landolt, rings with square gaps in different sectors. But their recognition is subject to great individual variations, and the same applies to Hess's international test-boards, in which the figures 0, 1, 4, and 7 are supposedly as difficult to recognize as Snellen's hooks are, combined with Landolt's rings.

Another point is that the average vision, normal and healthy eyes, is often considerably greater than "5-5," based upon the normal visual angle of 1'.

Purely physical visual tests are of equal value, lineal figures, for instance, being more difficult to distinguish than round ones. In addi-

tion, the size of the pupil also plays a part. Consequently, in corneal astigmatism of pathological degree and other refractive errors of the eye, a much higher central vision is found in contracted pupils than in dilated ones. All of which shows that it is not easy to establish a satisfactory method of testing the central vision.

Löhlein and Gebb have compiled a test-board containing figures, letters, and signs which presupposes a greater emmetropic average acuity than the ordinary test-boards. One row contains only test objects which "with uniform artificial illumination" have been recognizable for normal eyes with about equal facility. On the back of the board, school tests (hooks and signs) have been provided, with directions for teachers, by Steiger. They serve at the same time to determine visual acuity and the minimum degree of illumination required for reading, writing, sewing, drawing, and similar occupations.

The international normal vision tests of Wolffberg and the picture book illustrated by half-black and half-white pictures, which he compiled, after Leporello, to test the visual acuity of children, have been designed to suit the conceptions of basis and index, introduced by Wolffberg. The *basis* is formed by a desired figure of any size,—say Landolt's ring for instance,—which, however, must be constantly retained. The recognition of the basis does not serve as a measure of the visual acuity; this devolves upon the index, which, in Landolt's rings, is represented by the gaps.

The degree of visual acuity is expressed by the formula $V. c. (visus centralis) = \frac{D}{d}$ either in ordinary fraction, as for example: $V. c. 5/50$, $5/25$, $5/20$, etc., or by the decimal system: 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8, 0.9, 1.0, 1.5, 2.0, 2.5, 3.0, etc.

The higher grades of refraction errors, especially of hypermetropia and astigmatism, occur more frequently in imbeciles and idiots than in normal children.

The relations between hypermetropia or astigmatism and headache likewise deserve consideration. The pains are located, as a rule, in the frontal region above the orbits, sometimes, however, in the occipital region. The ciliary muscle is relaxed during sleep, but upon awakening is immediately under tension. This causes headache, which decreases after partaking of food and may disappear for the rest of the day, unless the eye is used for near work. These complaints disappear instantaneously upon paralyzing the ciliary muscle by cycloplegics, even in the presence of very low grades of refractive errors which require refractive correction.

Trousseau observed disturbance of sleep in children who had errors of refraction or had overexerted their eyes. The insomnia was often preceded by migraine or headache; the children could either not sleep at

all or not until the early morning hours, and then awoke with headache. Contributing factors are nervousness, psychic excitation, mental overwork, various diseases, especially the acute exanthemata. Correction of the refractive errors produces a return of normal sleep.

The glasses mostly in use are the spherical biconvex and biconcave. As the line of vision in various directions is to pass through the glass in a nearly vertical direction, periscopic lenses are preferable for correct vision.

The glasses invented by Franklin have two parts, the upper for distance and the lower for near work, both held in position by the rims. This has been improved upon by the unbifolux double focus lens, in which the distant and near segments are fused together, obliterating the dividing line between the two. Both parts are correctly centred and so improved for grinding as to do away with color and prismatic disturbances.

The cataract glasses with invisible segments, "Unibifoluxe-Lenticulare," weigh only one-third as much as ordinary cataract glasses. As these glasses are ground on a half curve, the field of vision is considerably larger than with ordinary cataract glasses.

There are also strongly curved glasses designated as "Engeem-nisken"; simple or compound cylindrical glasses, periscopic in shape; the so-called "toric" lenses for the correction of regular astigmatism, and of the telescopic spectacles devised by Hertel and so improved by Scott, of London, "that they give rise to much less aberration," look like and weigh no more than ordinary spectacles.

The Gullstrand-v. Rohr cataract glasses, manufactured by Carl Zeiss, of Jena, correct postoperative corneal astigmatism, which often nullifies the results of an otherwise successful operation. They are accurately made reading lenses which enable the wavering retina to receive minute images even at a considerable angle.

In order to see correctly through the centre of the lenses, the shape of the spectacle bridge should be adapted to the bridge of the nose, and there should be correct distance between the optical centres, the so-called pupillary distance. The measurements are made with a millimetre scale or the pupillary distance measure (Horstmann, Leplat, Maddox, etc.). When measuring the pupillary distance for distant vision the visual axes should be parallel, but when estimated for near use the visual lines should converge according to the type of near work required. When the pupillary distance is measured for with distant and near points the medium convergence of the two is taken. The prismatic effect of the edges of spherical glasses is made use of by decentering a concave lens outward and a convex lens inward, giving the same effect as that of a prism base in. This facilitates near work by reducing

the convergence of the visual lines which is usually associated with accommodative effort. For this reason, and especially when there is weakness of the internal rectus muscle (exophoria), concave glasses are selected in which the pupillary distance is somewhat greater than the actual pupillary distance, while in the case of convex lenses the measurement is correspondingly narrower.

1. HYPERMETROPIA

Hypermetropia is the most frequent refractive defect in the first years of life. It is caused by a relatively short eye, less often by flattening of the cornea or absence of the lens (axial or refractive hypermetropia). By exerting the accommodation, the child is capable of neutralizing, or masking, the hypermetropia for a time. Hypermetropia will remain latent until there is a physiological decrease in the range of accommodation. Hypermetropia is called *facultative* if the test for distance reveals equally good central vision with or without convex glasses; *absolute*, if visual acuity is reduced and must be correspondingly raised by convex glasses. Under certain conditions, owing to relaxation of accommodation for the near point, which is increased with convergence, fine print can still be read without the aid of a convex lens. Again, manifest hypermetropia is less in the monocular than in the binocular test, because the ciliary muscle is less tense when each eye is tested separately. While slight or medium degrees of hypermetropia do not, as a rule, require correction until about the twentieth year, there will be evidences of accommodative asthenopia in the higher degrees. This is especially true if there is pathological corneal astigmatism and the patient is doing a great deal of near work. These complaints for the most part consist of pressure in and over the eyes, which is likely to become a headache on the following day. This is due to the transmission of the ocular irritation through the ramus recurrens of the trigeminal nerve. The headache varies in proportion to the lack of rest, and can be removed only by proper convex glasses or sphericocylindrical lenses if regular or mixed astigmatism exists.

For relative hypermetropia, compare section on "Strabismus."

Treatment.—If in axial and refractive hypermetropia the latter condition is caused by flattening of the cornea, and near work causes complaints, the manifest hypermetropia should be corrected. This should be determined by binocular tests for the distant point. The glasses prescribed should be worn continually. They are also used for distant vision, if this affords considerable improvement. As a rule, stronger convex glasses for near work are only necessary when accommodation is impaired. Individuals with anisotropia, when using one eye only for sharp vision, take a stronger convex glass for near work,

because this will not cause the amblyopic eye to converge and, consequently, the relation between accommodation and convergence is not as intimate as in binocular vision.

In aphakia, one pair of glasses is required for distance and another for near use, for an aphakic eye has no power of accommodation. If the eye was previously emmetropic the required lens will be a 10 or 11 D. or correspondingly stronger in preëxisting ametropia. The nearer the object to be seen the stronger must be the lenses.

For example, a child whose eyes have been well corrected for distance with 11 D. sph. uses for reading at a distance of 25 cm. 15 D. sph. (11 D. and 4 D.: $4 = 25$ cm. focal width), etc. If the correction should be insufficient, improvement should be attempted by combinations with cylinders.

2. MYOPIA

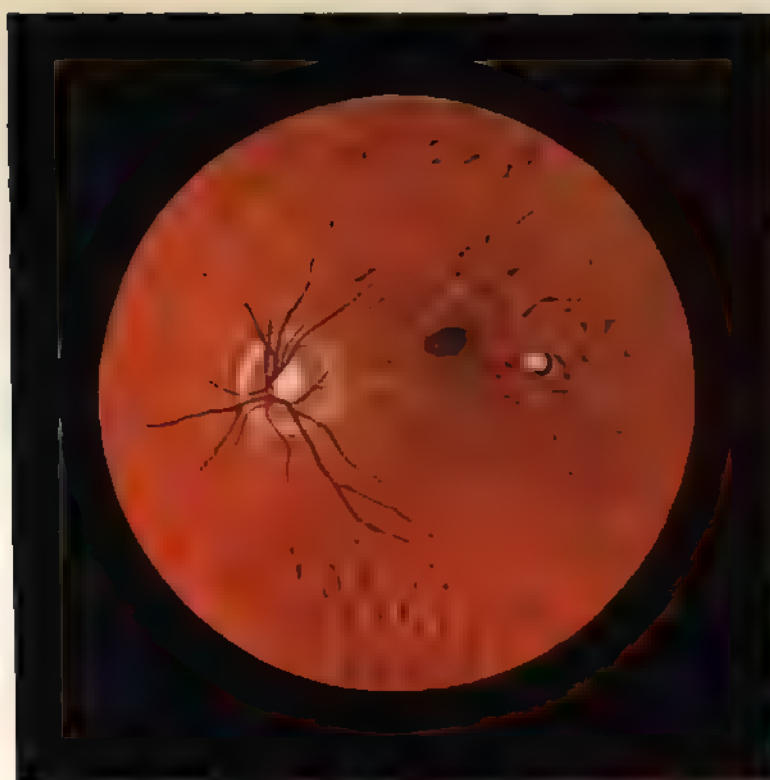
By typical (axial) myopia is meant the longitudinal structure of the eye as distinguished from the rarer corneal or lenticular refractive myopia. It begins either in the first years of life (myopia with posterior staphyloma) or it develops during school years, culminating in myopia of about six diopters (the so-called school or stationary myopia). As a rule, myopia with posterior staphyloma at once exceeds 6 D., is progressive, and visual function is markedly impaired by circumpapillary and macular (central) chorioretinitis (Plate XVIII, Fig. 1) and possibly by retinal detachment; on the other hand, in school myopia there are usually no ophthalmoscopic changes except for an annular or falciform conus of the disc margin (Plate XVIII, Fig. 2). Central vision is often very much less acute than in the normal eye. It decreases, according to Seggel, with the degree of myopia, so that, for example, in myopia of 6 to 6.75 D. the average visual acuity is no greater than 0.59 of the normal. According to the same author, the light sense undergoes impairment with the beginning of the myopic process, which, generally speaking, increases with the degree of myopia and is permanent at high degrees. Myopia may also develop later. Wirtz, for instance, observed myopia of right-11 D., left-13 D., in a twenty-eight-year-old saddler who had been in business from his fourteenth year and was enlisted in the marine service with full unaided visual acuity.

The transitory myopia in diabetes should not be overlooked.

The original causes of acquired myopia are still under dispute, but, aside from an hereditary predisposition, it is usually the direct consequence of near work.

While young girls are more inclined to myopia than boys, the latter showed an increased percentage under years of strenuous near work. As women take up scientific study and allied occupations, the reverse ratio bids fair to soon prevail.

PLATE XVIII.



Central chorioretinitis.



Oblique position of the opticus, temporal conus (myopia).

Full corrective glasses arrest school myopia to a certain degree, but it is not yet determined how near work changes the longitudinal structure of the eye.

According to the investigations of Hess and Heine, it is improbable that persistent tension of accommodation or contraction of the ciliary muscle causes increase of intra-ocular pressure, or that chorioidal tension causes pulling of the chorioid and subsequent enlargement of the growing eye. Far-sighted individuals with still greater tension of accommodation do not become short-sighted. Nevertheless, Best believes that increased tension of the chorioid by contractions of the ciliary muscle causes increased growth of the chorioid and sclera by irritation. The outer muscles, notably the internal rectus muscles, are said to be able to increase the intra-ocular pressure by impeded blood drainage (Arlt). But when the internal rectus muscle contracts it pulls away from the globe, while its antagonist relaxes correspondingly (Schnabel, Sherrington, Topolanski). According to Javal and Torner, the interrupted muscular contractions, such as occur in reading and writing, are injurious. Halben thinks that the malformation of the growing eye is caused by the increased pressure exerted by the external muscular tension; in convergence the posterior pole is principally affected. It must be remembered that the wall of the posterior section of the globe is not equal to that of the anterior section, the latter being made more resistant by tendons, muscles and tension of tissue elements.

Zirm adheres to the opinion that pressure of the internal recti is the cause of myopia, and explains that the sclera on the temporal side and retina are indirectly strained owing to convergence and to the fact that the optic disc is relatively fixed.

According to Elschning, myopia is not the result of dragging upon the optic nerve, because, according to his anatomical findings, the optic nerve in myopia is compressed and sinuous in the shape of the letter "S."

According to Stilling, the essential part of the myopic process is growth of the globe under muscular pressure. The tension of the superior oblique muscle is effected under simultaneous contraction of the lateral muscles, such as is necessary in near work, particularly in reading and writing; and this effort causes a longitudinal growth of the eye.

While the recti muscles, owing to their arrangement, cannot exert any particular pressure upon the globe, the pressure of two oblique muscles upon it is greater the lower the orbit. This is particularly true of the superior oblique muscle, the insertion of which is then lowered owing to the low position of the upper orbital margin, and its tendon will then reach so far back as to form a "closed loop."

Stilling mentions as an analogous example Indian children, whose skull, by bandaging, has been forced to grow upward in a steeple-like

form (oxycephalia). Ask confirms to a certain extent Stilling's statement on the relation of myopia to the structure of the eye, so far as the Swedes are concerned. Hamburger states that the closed loop mentioned by Stilling as being formed by the action of the superior oblique muscle is a post-mortem manifestation. Seppel, on the other hand, emphasizes the fact that the orbital structure is not the only factor in the production of myopia. Lohmann mentions the case of a man with a broad temporal conus, as a sign of acquired unilateral increase of refraction, while no external asymmetry of the face was recognizable, and the fronto-dorsal X-ray picture, taken during life, showed no difference between the right and left sides. While Heine and Römer consider weakness of the posterior scleral half the most important factor in the development of myopia, Stilling maintains that the sclera yields only in excessive myopia.

According to measurements taken by Gallus, this author claims that the structure and shape of the orbit are mainly responsible for the form of the globe. While in normal growth the globe extends forward, in myopia it is embedded deeper in the orbit. Consequently, myopic globes are more exposed to muscular pressure, thus causing not only elongation of the axis, but also greater corneal curvature.

While in other refractive abnormalities the orbits are short and bulky, the myopic type is long and small. Near work is a causative factor because strenuous reading and writing interferes with the normal position of the globe.

Danziger, too, looks upon certain conditions of the skull and orbit as influencing the refraction, based upon his skull measurements, made chiefly in deaf-mutes. "The myopic globe is long and low, because the skull and orbit are long."

Lohmann attributes the structural changes in myopia as possibly due to inherent developmental tendencies which produce disturbances in and variations of the normal structures. Even under normal conditions discrepancies appear. In the new-born the distance between the optic disc and macula is the same as in the adult, the measurement remaining the same, although the globe enlarges.

According to Kuschel, the increased refraction due to elongation of the axis is caused both by a congenital weakness of the corneoscleral tissue which it shares with all connective tissues of the body, and also as the result of continuous stretching of the posterior bulbar wall by backward pressure of the vitreous, occasioned by persistent use of the accommodation. This conception, which places the individual constitution in the foreground, is, in my opinion, the most correct one. I stated as far back as 1896 that a healthy eye in a healthy body cannot become short-sighted, even when undergoing the greatest efforts. At the same time, myopic eyes are abnormal, and as such have a lower average visual acuity than normal ones.

L. Weiss has described an arcuate strip with a silvery sheen running inward or slightly inward and downward from the disc. It can be seen by placing before the examined eye correcting lenses which are too weak. If stronger concave lenses are used the arc becomes less distinct in proportion as the details of the ocular fundus become more distinct, until finally it disappears. This observation is of importance, because it is also found in emmetropic and hypermetropic eyes. Its presence points to increased refraction due to ectasia of the posterior pole and an axial elongation and, consequently, to incipient or progressive myopia. The "myopic" changes of the optic disc are: On the nasal side, drawing over of the chorioid and apparent approach of the insertion of the central vessels to the margin of the disc; and on the temporal side, displacement of the pupil outward or outward and downward, widening of the scleral ring outward, and a curved displacement of the pigment line from the margin of the disc with the inclusion of a lighter halo of all shades running to the large conus, in which several sections can be recognized.

In a case of progressive myopia L. Weiss observed a displacement of the arcuate line which he assumed to be a reflex at the border of two optically different media, due to a discontinuity between retina and vitreous at the posterior end and an accumulation of fluid or detachment of the vitreous from the disc. The arcuate line receded forward and inward in such a way that with greater myopia the difference of refraction between focussing the retinal surface and focussing the place of the greatest distinctness of the arc amounted to 5 D. and more. Sometimes the arc again became less distinct, when the distance was further increased, until it finally disappeared as the myopia further increased.

This is not to be confused with the reflex observed by R. Berlin in hypermetropia, principally in hypermetropic astigmatism of children, and quite exceptionally in myopia. These reflexes often have the shape of a ring, lie directly in the region of the posterior lenticular surface, and still deeper when atropine is instilled. This phenomenon probably depends upon accommodation. "Sometimes two such reflexes are visible, which seem to lie closely side by side. If the head is slightly moved, the rings are seen to assume a parallax displacement against each other, the posterior one appearing as a shadow which the anterior one has thrown upon the retina."

An apparently slightly myopic eye may show ophthalmoscopic changes identical with those in one highly myopic. On the other hand, even in the highest degrees of juvenile myopia there may be no conus, or the chorioid may be somewhat abnormally pigmented at that place, while the anterior parts usually inaccessible to the ophthalmoscope will atrophy. H. Magnus called attention to ophthalmoscopically visible foci of degeneration at the border between the chorioid and the pars

plana of the ciliary body. Detachment of the retina has been observed not only in high degrees of myopia, but also in medium degrees. Although the latter condition is rare, usually detachment is of sudden onset. It is induced by congestions of the head, or any cause which tends to increase the blood-pressure suddenly, especially of the pulmonic circulation. The ophthalmoscopic diagnosis is not always easy in the early stages.

A permanent cure is rare, although spontaneous cures have occurred. Sometimes, by maintaining existing conditions, a relative improvement is brought about, but in most cases the original partial detachment of the retina becomes total. This may also be followed by insidious iridochorioiditis. In primary traumatic detachment, or if, as in exudative chorioretinitis, no tear of the retina has occurred, it usually becomes reattached.

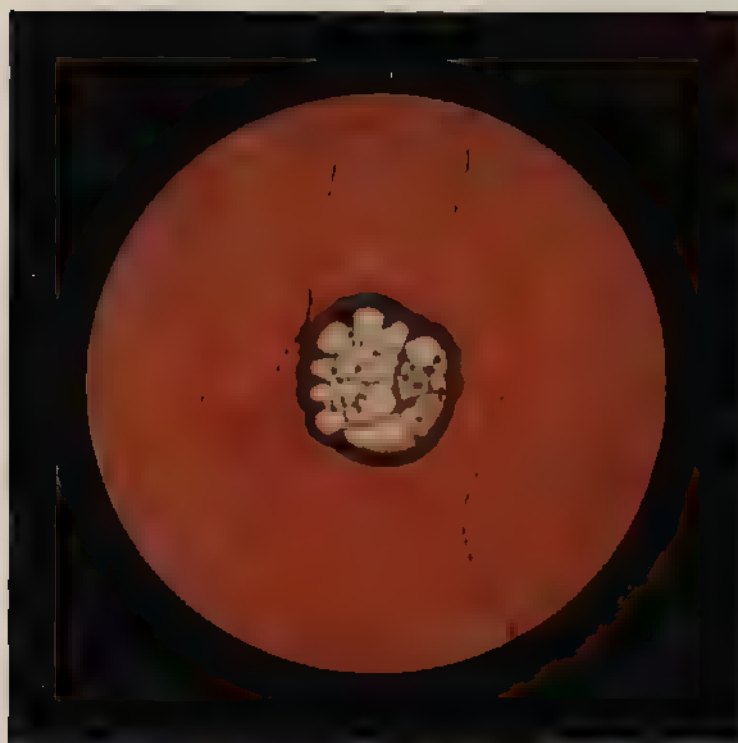
One case which healed spontaneously was caused by a hemorrhage from a large chorioid vessel. This may have been due to sudden increase in the blood-pressure, as the patient had taken a long, rapid walk on a hot day.

Treatment.—In order to prevent and improve myopia of whatever form, it is of prime importance that all near work should be carefully regulated. These children should be much in the open air and be instructed to practise distant vision. Well-lighted and ventilated rooms, at school and at home, and a good quality of reading and writing materials, are important factors. Physical and mental hygiene adapted to the individuality of the child, and in general a free and healthy mode of living, so that development and education may proceed along the proper channels, are most important. Everything that may cause congestions to the head,—cold hands and feet, constipation, impeded circulation by lacing or tight collars,—is to be avoided. The diet should be non-irritating. During convalescence from serious disease, the eyes must not be taxed until a return to health is completely assured. These measures for the prevention of myopia exert a distinctly favorable influence upon the general constitution, personal bearing, respiration, and circulation; they are the more important if there is an hereditary tendency to myopia.

The appointment of school physicians for high schools is very needful, as was repeatedly emphasized by E. Doernberger and others; this would ensure a careful examination of the pupil's eyes upon entering and leaving school, and should include a practical talk with the relatives regarding the health of the child and suitable occupation for him.

The so-called optometric subjective determination of the degree of myopia is made first monocularly and then binocularly. The youthful eye has a tendency to accommodative spasm (hypertonus; Pflanz), so

PLATE XIX.



So-called "coroboma" of the macula lutea.

that emmetropia and hypermetropia may either simulate or occasion myopia, or intensify an actual myopia which already exists. On the other hand, the reducing effect of strong concave glasses is such that in testing subjectively myopia may appear less than it really is, especially when associated with impaired central vision. At the same time, a weaker concave glass is pleasanter than one that completely corrects the high myopia, because it throws a larger, although less distinct, picture upon the retina.

The weakest glass for myopia is 2 to 6 D., which admits of the best binocular distant vision. These lenses are worn continually. A suitable correction which takes care of any existing astigmatism will, when worn constantly, sometimes arrest the progress of myopia or materially modify its course. In the slighter degrees with normal or nearly normal vision the finest print can be read with them without any effort. They will also reduce the asthenopic symptoms in near work which sometimes occur in slight myopia. The favorable effect of the so-called full correction is no doubt in a large measure explained by the fact that any disproportion between the balance of accommodation and convergence disappears. This prevents the close approach of the eyes to near work and also the bending of the head forward, which in the growing period is apt to cause hyperæmia of the eyes. Should pathological astigmatism be present, its careful correction is very important in order to improve the central vision for both the distant and near points, thereby enabling the child to work without unnecessary effort. To place a child with slight myopia on the front seat in order to make his work easier is a makeshift which should be resorted to only in cases which cannot be improved.

In higher degrees of myopia, however, full correction is not always borne well, so that, aside from fully or nearly fully correcting glasses for distance, a weaker pair may be required for near, enabling its wearer to read the smallest print of the "near" test type without effort at a distance of at least 20 to 25 cm.

The same method is adopted, so far as possible, for the highest grades of myopia. It is then very often seen that the best glass is one for partial correction only, and the glasses for near vision should be those most convenient for great distances also. Any disparity of the eyes (anisometropia or difference of refraction) should be compensated at an early period, so far as possible, by exercise with correcting glasses to attain binocular vision. Thus it may occur that glasses having greatly different degrees of correction are well borne. In obscure cases a specialist should be consulted, especially in progressive myopia and when there are complications with regular or irregular astigmatism, corneal opacities, etc.

He should decide also whether telescopic spectacles (Hertel) should be advised, or removal of the lens, as proposed by Fukala, for the improvement of the highest degrees of myopia (between 16 and 20 D.). It is advisable to operate on one eye only. Adams and others rightly stipulate that in cases for operation the macular region must be intact in both eyes, except for small irregularities of pigmentation; that the myopia must not be too far advanced; that the patient must be able to read the finest print without glasses, or with glasses the central vision must be equal to $1/4$ – $1/3$. Careful operators limit this operation to resection and paracentesis under the most careful aseptic precautions and manipulations, scrupulously avoiding injury to the vitreous. Should there later be an opacity of the pupil, due to proliferation of lens epithelium, which is not infrequent, the operation may have to be repeated; this, however, calls for the greatest caution and possibly general anæsthesia. There is danger of degeneration of the vitreous. In spite of all precautions, detachment of the retina has been repeatedly observed, even after the lapse of years.

Refraction myopia, due to disturbances in the refractive index of the lens incident to changes in form, or injury, demands, as far as possible, correction by lenses. Increase in the curvature of the cornea due to stationary keratoconus (compare p. 224), or keratoglobus or keratectasia, which are no longer amenable to medical or operative treatment, should be given such help as is possible with glasses.

The proper correction of short-sightedness is dependent upon repeated conscientious subjective tests, which may have to be supplemented by objective tests. The determination of refraction by the ophthalmoscope has the drawback that the foveal region cannot be utilized, owing to the invisibility of details. Atropine instillation allows parts of the cornea to participate in vision which would otherwise be excluded.

The dividing line between "lower," benign, stationary, transitory, progressive or school myopia, and "higher," malignant, permanently progressive or excessive myopia, is not determined by a certain degree in correcting lenses. School myopia may assume higher degrees than are ordinarily observed. It has been found that the pathological manifestations of excessive and malignant myopia, probably due to a congenital arrest of development, occur relatively often in individuals who read and write little or none. The myopia is fully developed before school age and progresses to the end of life.

The fundus of such eyes is often poorly pigmented at a very early age. At the edge of the optic disc may be seen a rather large falciform or irregular formation or ectasia in the posterior section of the globe (staphyloma posticum) with tears in the region of the chorioidal macula,

together with spots of retinitis and chorioretinitis. Highly myopic eyes are susceptible to hemorrhages from the retina and chorioidal vessels and to detachment of the retina.

The treatment is, therefore, limited to the correction of the abnormal refraction by suitable lenses, so far as possible.

Acute central chorioretinitis is often accompanied by hemorrhages, metamorphopsia, and positive central and pericentral scotoma, but further experience is required to decide whether and how far surgical removal of the lens can arrest further progress. These cases should be referred to an eye clinic as early as possible.

The treatment of detached retina is generally very tedious and must be in the hands of an ophthalmologist. Widely different methods have been instituted with varying degrees of success and failure.

Electricity furnishes the best indirect illumination of a closed work-room. A soft diffused and not too intense Moore light, or the high wire gauze lamps enclosed in globes and connected with reflectors (Tantal or Wolfram) may be used. In this way the light may be distributed so evenly that there is no shadow. For instance, in drawing classes, indirect illumination may be so perfectly arranged that a circle on a horizontal drawing-board is hardly visible. Experience, however, has shown that several hours' work under these conditions greatly fatigues the eye, and this kind of illumination is therefore bad, the light being too indirect. Again, indirect illumination, reflected exclusively from the ceiling, is insufficient for work on drawing-boards, which, in practice, are usually almost perpendicular, since the illumination is considerably reduced by the oblique direction of the rays. A half-indirect illumination such as is planned in the Moore light installation has therefore been considered the most perfect. In all cases where the natural light from windows is excluded and for a time replaced by artificial light the latter should be arranged near the windows—as, for instance, against pillars—in order to retain the contrasts produced by natural light (W. Wedding).

With artificial light of 50-candle power, the eye is capable of working just as rapidly and without fatigue as in average daylight. This candle power should not be exceeded, as sharp contrasts between light and dark make great demands upon the eye, and the greater the power of a single light-source, the more uneven is the distribution and the poorer the effect. Persons for whom the 16-candle incandescent lamp is insufficient appreciate an increase in illuminating power up to sixty-four candles when approaching the light within twenty inches. The lights of warm tone—with a large percentage of long red and yellow waves—are preferable, and have in large measure replaced the greenish gas incandescent lamps.

The most economic and general illumination in private houses is the

petroleum lamp, having a power from a few to twenty or forty candles. But it is not yet possible to supply enough oxygen to the flame by natural draught to ensure perfect combustion, as in the Bunsen light. The simple petroleum incandescent light is therefore not to be recommended.

Visual disturbances in childhood resembling myopia may occur by auto-suggestion. These children may have neurotic tendencies. They may be pale and underdeveloped, or rapidly overgrown and lean, are usually very lively and restless, frequently irritable and easily excited, and often complain of photophobia and fatigue of the eyes. They are usually the most alert and best pupils. By observing short-sighted children wearing spectacles, they imitate their symptoms, and a desire to wear spectacles plus any slight refraction anomaly starts an auto-suggestion which may considerably exaggerate any myopia that may be present. Where objects are brought too near the eye, the proper distance of the near point can be effected by plane glasses. Auto-suggestion may be suspected when a strong concave glass corrects the visual acuity just as well as a weaker one or a plane glass. In some cases, a weak convex glass or even an empty spectacle frame will instantly raise this pseudo-amblyopia to full visual acuity. Some children suggest to themselves amblyopia for near subjects. Objective ophthalmoscopic and skiascopic determination of refraction, together with the other ophthalmoscopic findings are just as important as the systematic use of plane glasses for the subjective determination of refraction in school children.

Treatment.—Tonics are prescribed, school work is temporarily restricted, while psychic treatment is particularly important. The principal task of the latter is to remove or avoid anything which may lead the children to further imitations. Weak convex glasses are not permissible, unless there is accommodative asthenopia due to hypermetropia.

For excessive blinking, which is not rare in high degrees of myopia, the symptomatic use of zeozan water (see p. 131) is recommended.

3. ASTIGMATISM

The following explanations will summarize the general facts about corneal astigmatism.

The curvature of the normal corneal centre is somewhat less in the horizontal than in the vertical direction. Unless the difference amounts to more than 0.5 to 0.75 D., this curvature is compensated for in the youthful eye by a reverse curvature of the corresponding meridians of the lens. Congenital reverse curvature of the corneal centre (*astigmatismus corneæ perversus*) is rare. It is not neutralized by a reverse astigmatism of the lens, be it ever so slight. This also applies to reversed corneal astigmatism that develops by reversion of a regular, uncorrected astigmatism.

For the glass correction of regular corneal astigmatism with lenses the degree and meridians of which may change in the course of growing years there are the following possibilities:

In or near the horizontal meridian:

Hyperopia
Emmetropia
Hyperopia
Myopia
Hyperopia

In or near the vertical meridian:

Emmetropia = hyperopic astigmatism
Myopia = myopic astigmatism
Hyperopia weaker = comp. hyperopic astigmatism
Myopia stronger = comp. myopic astigmatism
Myopia = mixed astigmatism

As these types are often deviated from, the XI International Ophthalmological Congress regulated the designations of the meridians for the atypical cases in the following way: In astigmatism, the physician, facing the patient, measures the degree of astigmatism in the upper semicircle of the test spectacles, so that zero is at the horizontal line nasally at both sides, the measuring proceeding upward and temporarily to 180°.

When measured with the keratoscope an evenly rounded picture of the concentrically arranged black and white circles of the paper disc is not seen, but instead an oval one, whose long axis corresponds to the meridian of least curvature, the refraction of which is weaker.

In ophthalmoscopical examination, in the inverted image, the convex lens must be held close to the eye; the disc will be seen considerably enlarged in the meridian with weaker refraction. In the upright image it is but little enlarged in the same meridian.

As congenital changes of form of the transverse section of the optic nerve may simulate astigmatism, regular corneal astigmatism is not definitely established, unless at the examination in the reverse picture the disc changes its form when the position of the convex lens is changed. If, for example, the convex lens is not held obliquely but is in close apposition to the patient's eye, the disc appears transversely oval, but at greater distance assumes a roundish shape. Finally, when the focal point of the lens becomes longitudinally oval, it falls in or before the iris plane.

The optometric examination can be carried out very well with Snellen's ray figure. The astigmatic eye can see distinctly the lines which are parallel to the principal meridian. The ordinary reading test chart usually contains types by which the abnormal meridians can be found.

The objective measurement of the corneal curvature is now generally effected with the Javal-Schiötz ophthalmometer. The fact that the meridians of the cornea which are of particular importance can be demonstrated largely facilitates the determination of the glasses which are necessary for a correct compensation of regular astigmatism.

Regular astigmatism is much more rarely dependent upon an anomaly of curvature or oblique position of the lens, due to a defect of the zonule of Zinn.

Treatment.—The earlier the error of refraction is corrected, the better will the development and increase of asthenopia be prevented. There is often a difference of refraction in the two eyes. Regular astigmatism is occasionally accompanied by a congenitally lowered function of the macular region and by congenital anomalies in the interior of the eye,—such as irregular form of the optic disc, downward conus, punctiform opacities of the lens, remnants of the persisting pupillary membrane, and pigment changes in the fundus.

It also happens that cylindrical glasses which do not correspond to the ophthalmoscopic findings are found subjectively the best, in spite of the faulty position of the meridians. In such a case an attempt must be made to approach the axes gradually to their proper position. This is usually successful.

When there is irregular corneal astigmatism due to absence of a focal point on the one meridian, the keratoscopic and ophthalmometric pictures are distorted. Irregular astigmatism may also be caused by corneal opacities, partial displacement or congenital deformity of the lens. Or an enlarged pupil, due to iridectomy or congenital or traumatic irideremia, may call attention to the irregular curvature of the margins of the lens.

The consequent disturbance of vision may be lessened by tattooing the corneal spots, wearing stenopic spectacles, either cribriform or with a fissure, and the hydrodiascope, mentioned on p. 224.

The correcting glasses should be selected by an experienced ophthalmologist, as he should also test aphakic eyes in which, in spite of a clear pupil, no satisfactory vision can be obtained with spherical lenses. Coexisting corneal astigmatism is nearly always atypical in form and of a high grade. The correction of this astigmatism does not always succeed in producing vision in an otherwise normal aphakic eye, because there is often, in addition to the atypical astigmatism, an irregular corneal astigmatism which cannot be corrected.

If the astigmatic individual wishes to use an opera glass without his spectacles, the lens must be either specially ground or cylinder glasses must be inserted before them so that shifting of the axis is impossible.

Silex removed astigmatism of 6 D. in an eight-year-old boy by three sclerotomies, and others have reported on operative treatment of regular corneal astigmatism; but further experience with this form of treatment is needed.

4. DISTURBANCES OF ACCOMMODATION

Accommodation is the ability which the eye possesses of collecting light rays emanating from near or distant objects and bringing them to a focus upon the retina, thus producing a sharply demarcated picture.

This is brought about by a change in the optical focus of the lens. At the same time, objects located at various distances may be seen with approximate distinctness without change of accommodation, when their circles of dispersion are so small as not to be perceived by the retina. According to Listing, an eye accommodated for infinity with a pupillary width of 4 mm. can still sharply perceive objects at a distance of about twenty-five metres.

The distance between the far and near point permitting of distinct vision, or the range of accommodation, is larger when measured monocularly than binocularly, because the resulting convergence of the visual lines influences the degree of possible accommodation. The relation between convergence and accommodation, however, is not absolute. Test types can be recognized just as distinctly at a certain distance when the accommodation has been increased by weak concave glasses or reduced by weak convex glasses.

5. PARALYSIS OF ACCOMMODATION

This "relative range of accommodation" depends more or less upon the convergence of the visual lines. Paralysis of accommodation must be suspected when an eye sees distinctly at a distance, but cannot read near type without glasses. This is diagnosed by determining the near and distant point of the eyes. There is complete paralysis of accommodation when the eye can read the finest writing only at a distance, at which it is focussed by corresponding convex glasses—an emmetropic eye, with + 4 D. str. at 25 cm.—and when the test types become indistinct when drawn nearer. On the other hand, the ciliary muscle is only paretic when the eye can still read the same matter at a slightly less distance. The accommodation which is still present is determined monocularly by looking for the near point of the eyes when aided with glasses.

For example, if an emmetropic eye, with normal visual acuity for distance, cannot read finest print, and with convex 4 it can still be read at 16 cm. with the greatest effort, then A (the range of accommodation) is equal to $\frac{100}{16}$ or 6.25 D. less 4 D., or equal to 2.25 D. The actual near point, therefore, is $\frac{100}{2.25}$ or 44.4 cm.

The cause may be a local toxæmia, due to cycloplegics. Like traumatic paralysis of accommodation due to contusions, etc., cycloplegia is associated with dilatation of the pupil (paralysis of the pupillary sphincter). Cycloplegia and iridoplegia, with sensory and atrophic disturbances of the eye, point to an affection of the ciliary ganglion, and, when combined with paralysis of the external muscles governed by the oculomotor nerve, to lesions of the oculomotor trunk. These lesions may be due to neuritis, tumors, gumma, solitary tubercle, basilar meningitis, basal fracture, or disseminated sclerosis of the brain and spinal

cord. Unilateral or bilateral nuclear paralysis of accommodation and pupil is occasioned by encephalitis, internal hydrocephalus, disseminated sclerosis, and acute infectious diseases. Post-diphtherial paralysis of accommodation is always bilateral. It also occurs in light abortive diphtherial angina, which either escapes observation or is mistaken for simple inflammation of the throat. At times the paralysis is accompanied by paresis of the pupillary sphincter. Disturbances of vision usually occur coincident with hypermetropia. The reduction of central vision observed in this condition is often deceptive, and will disappear by exact correction of refraction, and especially of rather pronounced corneal astigmatism. The prognosis of this type of paralysis is good, because it usually disappears under appropriate treatment in one or two months.

This, however, should not be confused with neurasthenic weakness of accommodation, which is often found in anæmia, connective with hysteria, neurasthenia, onanism, or during convalescence from grave febrile infectious diseases. It occurs in conjunction with irritation of the trigeminus and fatigue of the retina, and is especially noticeable when an object is held very near the eye. Children may be incapacitated for all near work for a short time, usually for one or two months and occasionally longer. The patients complain of cloudy vision, dull, pressing or boring pains in or behind the eye, occasionally in the forehead, head or nape of the neck, heaviness of the lids, burning and dryness of the eyes, sensitiveness to light. These symptoms may be absent, slight, partly or wholly present, when the eyes are at complete rest. Unlike muscular asthenopia, due to insufficiency of the internal or external recti muscles, or to hyperphoria (upward squinting), in high grades of myopia, or improperly corrected hyperopia anomyopia, the attempt to correct the disturbance in accommodative asthenopia by prismatic or convex glasses is doomed to failure. Furthermore, the complaints may change without corresponding ocular change, except anæmia or slight hyperæmia of the conjunctiva.

Weakness and paralysis of accommodation may be the first sign of increasing diabetes, which often causes a disproportionately rapid decrease of the absolute and relative range of accommodation, so that in hypermetropia the convex glasses required for near work must be exchanged for stronger ones at an unusually rapid rate. As the metabolic anomaly improves, these manifestations again decrease.

This "symptomatic" weakness of accommodation will also occur with increased weakness of the internal rectus muscle in nephritic children who have suffered a great loss of albumin, and also in chronic affections of the eyes, nose, and accessory sinuses. They are particularly pronounced when the eyes are hypermetropic.

Paralysis of accommodation as a late manifestation of syphilis occurs suddenly, and may rapidly disappear and return; it is usually unilateral and associated with paralysis of the pupillary sphincter. A cure is rare, if instillations of physostigmine have no effect upon the pupil and ciliary muscle.

In spasm of accommodation occurring after influenza, pertussis or exaggerated efforts, or as a sequel to supra- and infra-orbital neuralgia in anæmic and neurasthenic children, there is a restricted field of vision and a reduction in visual acuity for distance, which, unlike actual myopia, is not improved by concave glasses to a degree corresponding to their strength. Furthermore, the ophthalmoscopic and skiascopic examination of the refraction, even when accommodation is eliminated by a cycloplegia, demonstrated much less myopia or even emmetropia or hypermetropia. At the same time a careful general examination will reveal signs of anæmia or of neuropathy, circumscribed anæsthetic places, etc.

In children who are aphakic, whose lenses were removed in infancy, and in congenital hyperopia of high degree, it often happens that the correcting convex glasses do not give sufficient visual acuity, so that the child prefers doing without them and bringing his eyes as closely as possible to the reading and writing matter. This means seeing in circles of dispersion. This is rendered possible by the fact that with the approach of the object the size of the retinal image increases more rapidly than the size of the circles of dispersion. While Donders, Sattler, Hess, and Wagenmann deny that aphakic eyes possess accommodation, Fürst states that there is more or less accommodation in these individuals, so that sometimes the entire high-grade hypermetropia of the emmetropic aphakic eye (12-13 D.) may be compensated for both in the distant and near.

Fürst explains that this is not a pseudo-accommodation by astigmatism, nor shifting of the cataract spectacles, nor vision in circles of dispersion, but an increase in refraction. The accommodation is brought about by pressure of the orbicularis and the external muscles. There is an elevation of the index of the refracting media and partial regeneration of the lens, whose functioning elements are advanced into the pupillary region by the external muscular pressure.

The *treatment* in paralysis of accommodation depends upon the underlying cause. Temporary wearing of glasses may be considered, if the sight is impaired for near work. The strength of these glasses depends upon the degree of paralysis, the presence or absence of ametropia, and the average distance at which the work must be done. In hypermetropia a stronger glass is required than in emmetropia; in myopia a weaker one, or none at all, if the distant point of the myopic

eye coincides with the required working distance. As improvement progresses, the glasses are to be replaced by weaker ones or discarded altogether. In paresis of accommodation, which is very rarely unilateral, no optic correction is attempted, unless it facilitates work. Owing to the synergistic relation between accommodation and convergence, the distance of the centres of the glasses is preferably slightly less than the pupillary distance. If the course is more obstinate, tincture nux vomica is recommended, two to five drops to be taken internally once or twice a day, according to conditions.

Irritation of the ciliary muscle by weak galvanic currents has been recommended; they are conducted into split electrodes and thence to the closed lids. Instillations of pilocarpine mur. (2 per cent.) or arecolin (brom. hydrate) may also be tried to improve ciliary contraction. The instillations are made in the evening just before retiring. This also holds good for the daily instillation of a 1 per cent. solution of atropine sulph. for four to six weeks to dilate the pupil and to remove the much rarer accommodative spasm. This treatment has a particularly favorable effect if this accommodative disturbance accompanies or precedes myopia, as it often does. In order to prevent persistent winking due to light irritation, medium dark spectacles are worn during this time. Mydriatics, however, are sometimes powerless, while general hygienic measures, such as rest, general and ocular rest, and recreation in the mountains or at the seaside, have a more favorable effect. Reported improvement following the application of very strong faradic currents is probably based upon suggestion.

An eleven-months-old child which had been operated upon for unilateral cataract preferred seeing at close range without the use of glasses. Henker and Wagenmann effected improvement of vision in the affected eye for both near and distant by a monocular prismatic telescope (Zeiss) and a sextuple telescopic loupe. It is advisable to use the instrument on a specially constructed desk, which allows for the proper distance between glass and book, and which has an arrangement for shifting the book. In this way the instrument need not be supported by the hand.

XIV. DISTURBANCES OF PUPILLARY MOVEMENTS

THESE disturbances are of so many types that here those points only can be discussed which concern diagnosis and treatment in infancy and childhood.

The pupils are contracted in infancy but gradually grow larger until they reach their maximum size between the ages of six and twenty. As a rule, they are narrower in hypermetropia and wider in myopia than normal. Physiological contraction and dilatation vary in different individuals. Compare also remarks on p. 57.

Examination of the pupillary function is best done, according to Bach, as follows: In a dark room and at a distance of about sixteen inches from the patient, the physician rapidly illuminates the right and left pupils alternately with a concave mirror, such as is ordinarily used in ophthalmoscopy. More exact methods are Haab's pupillary scale, the isochrome pupillary scale of Krusius, the binocular pupillometer of Ohm, and pupillometer of v. Pflugk.

As light-sources petroleum, gas (Argand burner), or a small electric incandescent light with a ground glass globe may be used. The light is placed slightly behind the patient's head. The patient must look at a distant point over the head of the physician, who sits in front of him, so that convergence and accommodation may be eliminated.

If the pupils are equal, the direct light-reaction of the left eye and the indirect consensual reaction of the right eye are determined by placing the light at the left of the patient, slightly forward, and illuminating the left eye at a distance of 2-2½ inches with a convex lens of 13-20 D.; the patient again looking at a distant point over the physician's head.

The cone of light is then suddenly directed into the eye, but in order not to increase the quantity of light falling into the right eye, the hand holding the lens proceeds from the temporal to the nasal side.

The direct reaction of the right eye and the indirect consensual reaction of the left eye are examined by turning the patient's head slightly to the left without changing the position of the lamp or of the patient.

If there is no prompt and free light-reaction, one eye of the patient is covered and the light-reaction of the other eye is tested by noiselessly reducing the light several times by means of a regulator just low enough to recognize the width of the pupil, after which the light is rapidly turned high again. Any remnant of the light-reaction still present is demonstrated by directly illuminating the macula lutea through an incandescent gas lamp placed before the eye at a distance of about ten

inches. The light is allowed to act for some time, because the reaction is at times very slow. The contraction of the pupils caused by firmly pressing the lids together, or by the mere intention to do so and the muscular effort to prevent it, must be eliminated as a source of error. Otherwise a light-reflex may be simulated or a bad minimum reaction may appear more favorable.

The indirect consensual reaction is in most cases as great as direct light-reaction, although there are exceptions to this rule. When the second eye is exposed to as strong a light as was applied to the first eye, both pupils will again contract. This has been called secondary reaction by Weiler. It causes a further contraction of the pupils by 0.2–0.8 mm., representing the sum total of irritation. The contraction is at first rapid, then slow, and is followed by a slight secondary dilatation.

The convergent contraction of the pupils is more extensive than the light-reaction in normal eyes. It is determined like the accommodative reaction of the pupil, the patient fixing his eye upon his own outstretched finger at a distance of twelve or sixteen inches and slowly drawing it toward him.

Hemianopic reflex deafness is a controversial point (Wernicke). After experimental investigations, Hess and others call it valueless. But in a case of hemianopic reflex deafness, which had been established during life, a lesion of the tract was found at autopsy. Although its demonstration is liable to many errors, Behr admits the importance of hemiopic pupillary reaction, in order to differentiate cortical or sub-cortical hemianopsia from tractus hemianopsia.

With the exceptions mentioned, the examination of the pupils is concluded if, with lateral illumination, there has been a prompt and extensive direct and indirect reaction, if with transmitted light the pupils remain equally large and if the visual acuity and ocular fundus were found to be normal.

Congenital inequality of the pupils of small degree occurs in healthy subjects. There may also be spastic mydriasis (due to irritation of the dilator) and paralytic mydriasis as well as spastic myosis. The latter is also associated with temporary contracture of the ciliary muscle (pseudo-myopia), as in epileptic, and less often in hysteric, paroxysms.

The pupillary rigidity in hysterical and epileptic paroxysms is absolute. The pupils are sometimes oval.

Old iritis and the influence of myotics and mydriatics must be eliminated. If there is suspicion of artificial dilatation of the pupils, the demonstration of hysterical stigmata is impossible.

When the pupils are unequal, a disturbance of the pupillary reflex tracts should be considered. Inequality may also occur in healthy persons as a consequence of uneven illumination.

Reflex deafness (amaurotic pupillary rigidity) is occasioned by optic atrophy due to direct injury of the nerve or of the optic tract. If the right eye is completely blind, it will neither react to light stimulus, nor will its fellow react by indirect consensual reaction. If, however, the left pupil is illuminated, it will not only react itself, but the right one will, too. Upon reducing the light, the right pupil will be found slightly wider. After a period of uniform illumination, this difference gradually disappears. The convergent reaction is undisturbed. A considerable reduction of the pupillomotor excitability is usually present in choked disc. In order to explain reflex deafness after injuries without any change of visual power, it has been assumed that there are pupillary fibres which alone were injured by the trauma; but up to the present there is no unassailable proof for this assumption. Amaurosis has also been observed as due to atrophy of the optic nerve, while the light-reflexes of the pupils were maintained.

In bilateral blindness, neither pupil reacts to light either directly or indirectly. In this condition they are usually dilated, as contrasted with the contraction in reflex pupillary rigidity. The latter is caused by a centrifugal disturbance of the constricting tract on the other side of the oculomotor nucleus, and is associated with amaurosis in no more than about twelve per cent. of the cases.

In absolute pupillary rigidity, caused by complete paralysis of the sphincter or by atropine instillation, direct or indirect reaction to light, convergent reaction and dilatation following sensitive and psychic irritations are absent. Its value for differential diagnosis is relatively small, as it may be due to a great number of causes. It occurs after general infections and intoxications, after injuries to the skull, in contusions of the eye producing laceration of the sphincter, lesions of the short ciliary nerves, of the ciliary ganglion, of the motor roots of the ganglion, of the oculomotor trunk, of the ganglion cells and of the sphincter nucleus. Paralysis of the sphincter, due to a lesion of the nucleus or trunk of the oculomotor, is of particularly frequent occurrence in syphilitic and other organic cerebral affections. The pupillary rigidity which is associated with paralysis of the ciliary muscle (*ophthalmoplegia interna*) is often observed in connection with syphilis. Atropine does not produce further dilatation of the pupils, but cocaine does, owing to irritation of the dilator. An instillation of physostigmine is effective, provided the lesion is located behind the ciliary ganglion; it is less so if it is located before or in the ganglion. It is less often due to an arrest of the sphincter tonus on the part of the cerebral cortex.

Reflex pupillary rigidity (Argyll-Robertson's sign) occurs unilaterally occasionally, and remains so for a considerable time. It is permanent and isolated principally in congenital or acquired syphilis or as

an early symptom of the tabes and paralysis, much less frequently in affections of the medulla oblongata and the pons. Direct and indirect reaction to light, the pupillary unrest which is always observed in healthy individuals, and the reaction to sensitive and psychic irritations are absent. One pupil is often more contracted than the other, usually the one in which the disturbance of innervation is most advanced. The pupils often lose their round shape, but they are not dilated. Convergent reaction is usually so well retained that in cases of doubt prompt and extensive convergent accommodative reaction points to reflex rigidity, while a lazy or less extensive one points to "incomplete absolute rigidity." The prodromal signs of reflex rigidity are limited movement of the iris under illumination, without impairment of the convergent reaction, slow onset of the light-reaction, rapid abatement of the sphincter tonus upon illumination of the retina, and absence of secondary light-reaction (Weiler). While in cerebral syphilis there may be all kinds of pupillary disturbances which are often associated with other disturbances of ocular muscles, reflex rigidity of the pupils is but rarely among them.

In tabes pupillary symptoms are frequently present for years before the signs of Westphal and Romberg can be observed. Their absence is a valuable differential factor against the hypochondriac form of neurasthenia (tabes illusoria); against rheumatico-neuralgic conditions, which are sometimes regarded as incipient tabes; and against acute ataxia and the so-called neurotabes peripherica and diabetica. Should the behavior of the pupils give rise to doubts, bladder disturbances (ischuria paradoxa) may be of diagnostic importance, as they are frequently early symptoms of genuine tabes.

Paralysis of the oculopupillary fibres of the sympathetic, the so-called Horner's symptom complex, is of general practical importance. This symptom complex may be clearly defined or somewhat obscure. It consists of myosis due to paralysis of the pupillary dilator, contraction of the palpebral fissure due to drooping of the upper lid and slight elevation of the lower lid, paralysis of the superior and inferior levators, exophthalmos, paralysis of the orbitalis muscle and hypotony due to changes of the vascular tension in the eye.

The direct and indirect pupil light-reflexes are present in most cases, although very feeble. Instillation of a 1 or 2 per cent. cocaine solution will not dilate the pupil, but a stronger solution of atropine will do so, owing to sphincter paralysis.

When the paralysis is fresh, the corresponding side of the face is often hyperæmic, while in paralysis of long standing it is paler than the other side. Perspiration is absent on the affected side, while on the other side it is stimulated. Gradually facial hemiatrophy will ensue.

Upon stimulation of the sympathetic, the pupil becomes dilated, the corresponding half of the face is pale and perspires more than the other. The reaction to light is affected only by a very strong stimulus.

These phenomena, which point to a compression of the eighth cervical and the first dorsal nerve and of the corresponding segments of the spinal cord, are of diagnostic importance for affections located in the lateral parts of the neck or in the highest part of the thorax and posterior mediastinum, enlarged cervical glands or tumors, goitre extending toward the lung, unilateral tuberculous affections of the apex of the lung, or pleurisy.

Pupillary reaction in the infectious diseases of childhood has different signs. It is present in varicella, measles and scarlet fever, more or less reduced or absent in variola. In typhoid fever it becomes lazy when complicated by a pulmonary affection. In infectious endocarditis it may be entirely absent; while it is never present in peritonitis. The pupils are narrow in the early stage, sometimes also at the climax of epidemic cerebrospinal meningitis. The transition in paralytic mydriasis points to a destruction of nerve-fibres and is therefore ominous. Unilateral dilatation points to oculomotor paralysis, and medium or great dilatation, and strikingly lazy or almost totally arrested reaction of both pupils, point to general cerebral paralysis and impending death.

The sudden maximum dilatation of the pupils in asphyxia is due to a paralysis of the centres of the autonomous oculomotor. When administering anæsthesia, the physician should note the contraction of the pupils.

Treatment.—Treatment in all pupillary disturbances is always dependent upon the cause. If there is a disturbing bilateral mydriasis, colored protective spectacles of various tints are prescribed; in the unilateral affection, perhaps, exclusion of the affected eye by an opaque covering. Palliative treatment consists of instillations of morphine-physostigmine and tinct. of opium. Pilocarpine is less desirable, as it will later lead to paresis of the terminal fibres of the oculomotor and, by displacing the near point outward, spasm of accommodation. Exercises of the eye at the near point with convex glasses, and induced and direct electric currents have also been tried.

XV. AFFECTIONS OF THE EXTERNAL MUSCLES OF THE EYE

I. ASTHENOPIA

THE most frequent cause of muscular asthenopia is insufficiency of the two internal rectus muscles in convergence.

Early fatigue of the eyes in near work, blurred vision of near objects, pressure, burning, lachrymation, frontal headache of varying degrees, pain even on looking at people during conversation, twitching of the lids, chronic irritation of the conjunctiva: these are signs of muscular asthenopia. To recognize this condition, the distance from the eye where convergence fails is measured, while an object, such as a finger drawn toward the nose, is seen double. A more exact determination of dynamic convergence, the convergent near point, is obtained with Landolt's ophthalmodynamometer. A shining point in a darkened room is slowly approached toward the child until two images are perceived. To test the muscular equilibrium of the eyes, or to determine whether there is merely latent outward squinting, one eye is covered and a finger is slowly approached to within 25 cm. of the centre of the face. It is then ascertained whether the covered eye converges and focuses in the same way, or at least nearly so, as the uncovered eye (orthophoria); or whether upon removal of the cover the eye turns inward.

Graefe's test is more exact. A spectacle frame containing one prism of 10° for one eye, with the base upward or downward, is used. The patient looks fixedly through it at a sheet of white paper at a distance of 25 or 30 cm., on which there is a black dot. A lighted candle may be used instead. If the two pictures produced by the prism do not stand precisely above each other, but are latterly displaced, and if a prism down to 3° , with the base in, does not make the two pictures stand vertically above each other, it is a case of dynamic (latent) squint or heterophoria, or, more particularly, dynamic divergent squinting exophoria, which deserves attention. The eyes are expected, without fatigue, to converge at a distance of about three times less than the average working distance. Thus, at 30 cm. working distance, the eyes should be able to converge at 10 cm., and so on.

The degree of heterophoria is determined by the degree of the prism which makes it disappear, or corrects it. The degree of deviation can be directly read off on Maddox's tangent scale, an instrument which also facilitates the equilibrium test.

Treatment.—In hypermetropia, convex glasses should be tried first, as they often give the desired prismatic effect by reducing the

distance between the centres of the lenses. If they do not, the required prism can be combined with them. In myopia, the reverse procedure is instituted by first increasing the distance between the centres, and afterwards making a prismatic combination, if necessary. In emmetropia, simple prisms are prescribed with bases in. As a rule, none but 3 to 4° are borne well, because stronger ones produce color dispersion and unevenness of the field of vision, so that adducent prisms, for instance, make a vertical plane appear as concave, and an adducent prism as convex. It often happens that children can be suited with weaker prisms than correspond to the mathematic measurement of the convergent defect. As anæmia and other constitutional weaknesses are often the cause of the motor disturbance, they should receive proper medical attention. It may be necessary to restore orthophoria by a surgical operation (lengthening the external rectus muscle or shortening the internal rectus muscle). For this procedure, however, only higher grades of latent squinting, say from 8 to 10°, are suitable.

Finally, it is noteworthy that the correction of this disturbance of balance as well as that of exophoria—weakness of the external rectus muscle, which occurs less often than the insufficiency of the internal rectus muscle—may under certain circumstances effect an improvement of localized nervous complaints and of the general constitution. Furthermore, even slight degrees of heterophoria may cause grave nervous manifestations, when the power of resistance is reduced by disease, fatigue, or a neurotic disposition. This is also true of the vertical deflection, disturbance of the vertical position of rest, and especially of the so-called upward squinting. (Compare below.)

2. CONCOMITANT SQUINT (STRABISMUS CONCOMITANS, CONVERGENS AND DIVERGENS)

A marked degree of strabismus can be recognized with the unaided eye from the fact that there is no central fixation.

An apparent strabismus which is due to an abnormal position of the facial line to the ocular axis can be excluded only by covering one eye and observing whether the other one makes a fixation movement. With a very great angle Y (angle between the facial line and ocular axis) there is an apparent divergence; with a positive angle Y there is an apparent pathological convergence of the visual axes, the facial lines being parallel. In order to find out whether the squinting is monolateral or alternating in both eyes (alternating strabismus), a hand is held before the patient, who is requested to fix it alternately in the right and left halves of the field of vision or, in upward squint, in the upper and lower halves.

If the same eye is invariably used for this purpose, the strabismus is monolateral. When the fixing eye is covered, forcing the squinting eye to fix, it will persist in this position, even after the other eye has been

liberated. Otherwise there is an alternating squint. In alternating concomitant strabismus the opposite eye squints in every visual lateral half, while in alternating divergent strabismus the eye of the same side will usually squint.

The degree of deviation in strabismus is determined by the extent of the fixation movement which occurs in the squinting eye when fixing objects at various distances, the other eye being covered.

The simplest way to make the measurement is with a millimetre rule, applied to the lower palpebral margin. Having alternately covered and uncovered the fixing eye, the number of millimetres can be read off by which the squinting eye deviates from the normal position. A more exact method consists in measuring the angle of squint, which means the angle formed by the facial line of the squinting eye with the direction it should take under normal conditions. This is done at the arc of the perimeter, on Maddox's tangent scale or with Krusius's disc-deviometer. By this method also vertical deviations may be estimated.

The relatively frequent occurrence of concomitant convergent strabismus in hypermetropia is explained in the following manner. Hypermetropic persons with lowered vision, in order to avoid seeing indistinctly with both eyes, procure a sharp retinal picture of an object on one eye by accommodating the same very strongly. As a result the internal rectus muscle, working synergistically with the ciliary muscle, contracts out of proportion to the distance from the object. This causes the eye to turn inward. This "accommodative squinting" in relative hypermetropia is at first only a periodical occurrence, but it also occurs as one symptom of fully developed concomitant convergent strabismus.

Monolateral divergent concomitant strabismus is often due to a complete exclusion of the squinting eye from the binocular vision in consequence of unilateral congenital or acquired amblyopia or amaurosis of the highest grade. Permanent divergent strabismus also often results from periodic or dynamic divergent squinting, which is a frequent concomitant manifestation of high-grade myopia.

The difference between concomitant and paralytic strabismus is this:

As a rule, in concomitant squinting there is no double vision.

In paralytic strabismus, due to an old paralysis of one or more external ocular muscles in which contracture of the antagonist or antagonists has taken place, and which is manifest when the eyes are at rest, the pathological eye will not return when looking in the field governed by the paralyzed muscle.

The "secondary" deviation of the normal eye, when the paralyzed eye fixes, is greater than the "primary" deviation of the paralyzed eye when the normal eye fixes. As a rule, both are alike in concomitant divergent and convergent strabismus without upward deflection.

In actual squinting, where concomitant strabismus has not existed for a long time, the squinting eye deviates more or less from the normal direction, but accompanies the healthy eye in all directions. The excursion of both eyes is therefore the same. The defect is distinctly visible in the fixation of an object.

Concomitant strabismus is not often congenital, except in irregular formation of the skull, in great disproportion in ocular equilibrium and in pathological changes of the eyes with reduced visual acuity.

Concomitant convergent strabismus is caused by abnormal insertion of the ocular muscles, hypermetropia, anisometropia, and defective development of the centres regulating fusion. The latter condition applies particularly to alternating strabismus, where the central vision of both

FIG. 38.



Convergent strabismus.

FIG. 39.



Divergent strabismus.

eyes seems to be alike or normal. Physically weak and mentally inferior or precocious children seem to have a greater tendency to its development than normally developed children. External causes are acute febrile diseases (pertussis, measles, scarlet fever, typhoid fever, etc.), violent psychic emotions and similar factors.

Monolateral squinting is nearly always accompanied by considerable asthenopia. It is either congenital or acquired from non-use of the eyes due to errors of refraction—in convergent strabismus, usually to hypermetropia or hypermetropic astigmatism; in divergent strabismus, chiefly to high-grade myopia and myopic astigmatism. Amblyopia occurs as a consequence of corneal spots, opacities of the lens and vitreous, or incurable fundus diseases.

Concomitant convergent strabismus is divided into preliminary or truly periodical, constant, monocular, and accidentally alternating or

fundamentally alternating squint. Fixation in alternating strabismus is also due to anomalies of refraction and the muscular equilibrium, and occurs alternately in either eye. Coincident sursum or deorsum strabismus often disappears, as the inward and outward squint are cured.

Heredity is a notable factor. Congenital convergent strabismus, with unilateral functional irregularity of vision, occurs in 70 per cent. of the cases according to Jessen, in 33 per cent. according to H. Kohn. Worth found an hereditary history in 51.78 per cent.

Upward squint also occurs alone or congenitally on the same basis as lateral squinting, especially when the orbits are not level; it may also follow cicatrization in the orbit (after abscesses). The disturbance of binocular vision occasioned thereby interferes principally with the perception of horizontal lines, but may also cause persistent headache, an uneasy feeling of vertigo with sympathetic excitation of the vagus system and grave nervous symptoms of the stomach and heart. Upward squinting is particularly noticeable when the effort to unite the horizontal outline of objects with both eyes is not supported by vertical lines. It is, therefore, especially pronounced when travelling at sea.

Testing for vertical strabismus can be done on the plan of E. Miller. He uses two planes, 70 cm. square, which are traversed by parallel lines—one light with black lines and one red with green lines, the lines being 1 mm. thick and 8 mm. apart. When an upward squinter looks steadily at such a sheet, held horizontally just before his eyes, after a few seconds the lines will waver and dance. When the upward squint is compensated by a prism, the wavering of the lines disappears and the sheet may be looked at for any length of time with perfect ease.

In the same way insufficient convergence and its compensation by prisms can be demonstrated by holding the lines vertically.

The fact that pseudo-strabismus, which often occurs in infants during the first months of life, is also present in gastric and other disorders, has no significance.

The frequent occurrence of asthenopia of the squinting eye without recognizable changes is often a consequence, wholly or in part, of its non-use. It has often been observed that the visual acuity is impaired only after squinting commences and that it is distinctly improved after it is used in fixation. Again, the percentage of cases grows with amblyopia of the squinting eye, as well as the degree of the asthenopia with duration of squint. The younger a child the more easily and quickly can amblyopia be removed (Worth). Attempts have been made to improve asthenopic squint eyes by systematic daily exercise with reading glasses strong at first and made gradually weaker, with large type at first and progressively smaller, the healthy eye being covered. But material improvement in visual acuity rarely follows, because of the

impracticability of using the squinting eye exclusively for months and years for purposes of fixation.

Treatment.—Slight or periodic convergent, concomitant strabismus associated with hypermetropia, with one eye slightly impaired, is often corrected by the permanent wearing of suitable glasses. Slight or periodic myopia divergent, concomitant strabismus is also often cured by the early and exact correction of the myopia and myopic astigmatism. It may be necessary to combine the convex glass with an abducent prism, and the concave glass with an adducent one. Any deflection downward (hypophoria) or upward (hyperphoria) must also be considered. In upward squinting, especially, complete compensation, together with rapid and permanent removal of all nervous symptoms, is attained by a prism which may have to be combined with a glass correcting any refractive error.

The following operative measures used to correct anomalies of the position: Retroposition by resection of the tendon of the muscle to its bulbar insertion and looping of the severed tendon; advancement of the antagonist, or both steps together. To carry out these operations or to decide upon their exact indication and method of execution is exclusively within the province of an experienced ophthalmic surgeon. Even in their hands, and in spite of apparently favorable conditions and the greatest caution, success is mingled with failures. Certain anatomico-physiological anomalies, above all congenital insufficiency of fusion, or abnormal conditions of excitation in the oculomotor apparatus (Bielschowsky) contribute largely to unsuccessful results. The faulty position of the eyes may return; or after the operation there may be such persistent and annoying double vision that the patient wishes the former position of the eyes restored. Caution is particularly indicated in operative interference in neuropathic divergent strabismus. It has usually been present from early childhood, and may be a constant or periodical manifestation, monocular or alternating.

After the operation, continued exercises for vision and fusion, selection of the proper correcting glasses, maintaining the correct position of the eyes, and preventing recurrent strabismus are indispensable. It is also advisable to keep both eyes closed for four or five days after the operation, changing the bandage daily, even if but one eye has been operated upon; this facilitates the proper healing-in of the displaced muscle and may prevent a secondary operation.

A restoration of fusion power, sufficient to insure binocular vision, cannot always be attained, even with the best operative results; but the younger the child, the sooner will it be secured. This is true of stereoscopic vision also; but even there it is advisable, after the squint operation, to continue exercising with stereoscopic pictures (Dahlfeld, Kroll, etc.).

through direct affection of the nuclei of the ocular muscles or their supranuclear centres and tracts. The cerebral nerves may be directly injured through cranial trauma, notably in basal fracture, or may become damaged by secondary cerebral abscesses.

In primary or metastatic tumors of the brain and its meninges there are often, isolated or combined, paralyses and nystagmus. Congenital or acquired syphilis is a much rarer etiological factor.

For diagnostic purposes it is important to note that paralysis of the ocular muscles or of the facial nerve is often the only sign of tubercular tumor, especially of the pons or the corpora quadrigemina.

FIG. 40.



Paralysis of the right oculomotorius nerve since her third month, in 18-months-old child. No lues

Paralysis of vision, meaning the disturbed movement of associated muscles of both eyes, preventing both eyes from looking to the left, upward, etc., is due to central affections, notably of the region of the pons. Central disease is also responsible for paralysis of convergence and divergence, and the associated spasms of the ocular muscles known as "*déviation conjugées*."

Diagnosis.—To demonstrate paralysis of the lateral muscles in the eye of the new-born immediately after birth, M. Bartels uses the well-known rotatory nystagmus. Thus, if there is paralysis of the external rectus muscle of the left eye

and the child is constantly turned to the right with the head erect, the left eye will never go beyond the median line. The same effect is observed in half turns to the left. For older children, light anæsthesia is required in order to exclude nystagmus and fixation.

In any case, consultation with an ophthalmologist is advisable. Except the diagnosis of a pronounced abducent or oculomotor paralysis, the exact determination of other disturbances in the motility of the eyes by diplopia and other tests is not always a simple matter. This is specially true in secondary contracture of the antagonists. Diplopia is not always as easily noticed in children as in adults. There are cases of monocular psychogenic diplopia.

The most striking *symptoms* of the paralyses are defects of motility in that part of the field of vision governed by the paralyzed muscle, diplopia; of "false projection," facial vertigo disappearing upon closing

lids produces tenderness, followed by pains which radiate to the frontal and temporal regions. These and similar general muscular symptoms gradually decrease in the course of the disease and finally disappear.

Treatment.—For marked photophobia, dark gray spectacles should be worn. Frequent instillations of suprarenin-cocaine may have to be made.

Spasms of convergence, sometimes occurring in neurotic children, is a different affection from abducent paralysis. While in the former, as a rule, the ocular excursion is normal, during fixation only one eye will follow the object. In paralysis of the external rectus, excision of the globe outward is limited.

The treatment is causal.

4. PARALYSIS OF THE OCULAR MUSCLES

Depending upon the location of the lesion, paralyzes of the ocular muscles are divided into central, nuclear, or fascicular, and peripheral, basal, or orbital.

Plate XX gives a general survey of the topographical anatomy of the orbit and the base of the skull.

Although paralysis of the ocular muscles is very rare in infancy, there may be injury to the nuclei of the ocular muscles, especially at birth, the result of moulding or prolonged asphyxia; chronic, progressive ophthalmoplegia which appears early, but remains isolated and is not a disease pertaining exclusively to infancy.

Etiology.—The etiological factor in infancy is tuberculosis of the brain and its meninges. Tubercular cerebral tumors and basilar meningitis, by raising intracranial pressure, excite inflammation of the exposed cranial nerves. Enlarged tubercular, bronchial glands produce irritative lesions at the sympathetic, as shown by an inequality of the pupils, etc.

Other etiological factors are acute and chronic intoxications, auto-intoxications, either of intestinal origin or subsequent to primary and secondary renal diseases. Influenza and diphtheria usually produce paralysis of accommodation, without attacking other muscles. The secondary infections of the ear, such as otitis media occurring in the course of acute infectious diseases, also cause paralysis of the external ocular muscles. The transitory forms of paralysis in pertussis are due to stagnation, oedema and anæmia of the brain, while the persisting paralysis, especially that of vision, is nearly always due to cerebral hemorrhages. Paralysis following scarlet fever and measles is nearly always due to a secondary infection of the nose and accessory sinuses, meningitis, orbital phlegmons or sinus thrombosis.

The etiology also includes diabetes, cerebrospinal meningitis, and cerebral infantile paralysis. Infantile tabes is responsible for transitory paralyzes. Encephalitis in epidemic poliomyelitis produces paralyzes

PLATE XX

On the Right, the Nerves; on the Left, the Vessels of the Orbits.

AA. The eyeballs. The musculi levator palpebræ superioris and rectus superior are removed, also the supra-orbital nerve on the right.

BB. The medial cranial fossa. On the right the dura is completely removed. The osseous parts are broken away in order to show the muscles and nerves underneath.

C. The right half of the tentorium cerebelli is kept in moderate tension by the corresponding cerebellar hemisphere underneath, and is illustrated to show the course of the nervus tentorii.

C. On the left side the tentorium is partially dissected away, showing the cerebral nerves from the seventh to the twelfth, which are hidden on the right side.

I. Right olfactory nerve. The course of the ethmoidal nerve is represented on the lamina cribrosa.

II. The optic nerves, on the left with their sheaths, on the right, the nerve-trunk is exposed in the orbit.

III. The motor oculi in its course to the orbit, where it sends the short or motor root to the ciliary ganglion.

IV. Trochlear nerves slightly displaced medially from the first branch of the trigeminus, ramus primus nervi trigemini, in order to show the sympathetic root for the ophthalmic ganglion.

IVe. Insertion of the trochlear nerve in the superior oblique ocular muscle.

V. Trigeminus. The fifth root is surrounded by the tentorium cerebelli.

VI. Abducens nerve. The course of the nerve in the sinus cavernosus, its position in relation to the other nerves in the fissura orbitalis superior, and the insertion in the external rectus muscle are brought to view.

VII. Facial nerve of the left side which together with the

VIII. Acoustic auditory nerve enters into the porus acusticus internus.

IX. Glossopharyngeal nerve.

X. Vagus nerve.

XI. Accessory nerve.

XII. Hypoglossal nerves. The figure XII is on the resected medulla oblongata.

1. Gasserian ganglion.

2. The third branch of the trigeminus passes immediately after its origin through the foramen ovale.

3. The second branch, running through the medial cranial fossa anteriorly toward the foramen rotundum in order to proceed through the latter to the fossa sphenopalatina.

4. The first branch (the ophthalmic), from which the supra-orbital nerve has been dissected away.

5. Sympathetic root or trochica ganglii ciliaris.

6. Long root or sensitiva ganglii ciliaris.

7. Abducens nerve.

8. Short root or motoria ganglii ciliaris.

9. Ciliary or ophthalmic ganglion.

10. The short ciliary nerves emanating from the ganglion pass partly at the outer, partly at the lower side of the optic nerve anteriorly to the sclera, which is perforated by the ganglion. The short ciliary nerves, arriving underneath the optic nerve, fuse with the long ciliary nerves coming from the nasociliary nerve (12) and pass together through the inner and upper side through the sclera.

11. The lachrymal nerve, emanating from the first root, is embedded in the partly visible lachrymal gland.

12. Nasociliary nerve, having given off the long ciliary nerve and the infratrochlear nerve, passes underneath the internal oblique ocular muscle to the foramen ethmoidale and, giving off a few threads to the mucosa of the ethmoidal cells, passes through the foramen cribrosum to the nasal cavity.

13. Nervus tentorii, emanating from the first root, encircles with its two roots the trochlear nerve, takes a backward course, and is lost in the tentorium of the sinus transversus.

14. Supra-orbital nerve.

15i. Infratrochlear nerve.

15a. Supratrochlear nerve.

16. Lachrymal nerve, inserted with two branches.

17. Nervus subcutaneus malæ, visible in the deep-lying parts.

18. Fusion of the same with the nervus lacrimalis.

19. Nervus buccinatorius, passing through the musculus pterigoideus externus, and giving off a ramus temporalis at the outer side of the muscle.

20. External pterygoid nerve.

21. Nervi temporales profundi (anterior and posterior).

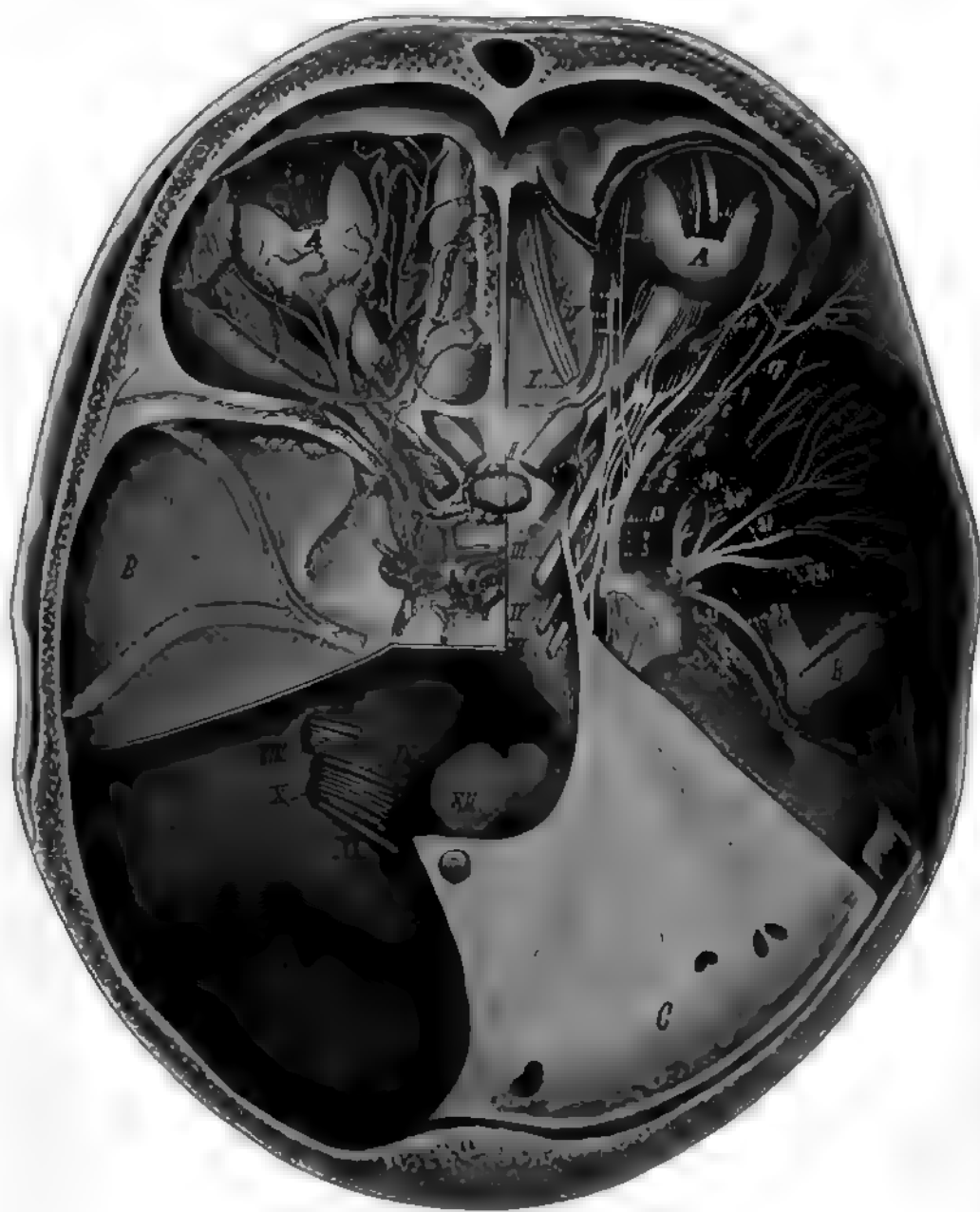
22. Nervus massetericus.

23. Nervus auriculo-temporalis.

24. Nervus petrosus superficialis major.

25. Weak nervus petrosus superficialis minor.

Ca.a., carotid interna; *a.o.*, art. ophthalmica; *e.e.a.*, vasa ethmoidalia ant.; *e.e.p.*, vasa ethmoidalia post.; *c.m.*, art. and vena meningea media; *S.c.*, sinus circularis; *S.ca.*, sinus cavernosus; *S.p.*, sinus petrosus superior; *P.b.*, plexus basilaris; *V.c.*, vena cerebri media; *V.o.m.*, van ophthalmo-meningea; *S.sph.*, sinus sphenoparietalis; *V.o.*, vena ophthalmica sup.; *V.i.*, vena ophthalmica inf.; *r.l.*, vena lacrimalis; *v.v.*, venæ vorticossæ.



the paralyzed eye; and, finally, the characteristic faulty head posture, which is assumed to correct the visual disturbance.

As to the differential diagnosis, the remarks made on p. 312 hold good here, with this addition: one of the reasons why examination of the secondary deviation is so important is that from it slight defects of motility, due to slight paresis, and their direction can be recognized.

The characteristic feature of cicatricial fixation of the globe, due to injury or orbital phlegmons, and which may resemble paralysis of a muscle, is the distinct retraction of the globe which takes place with maximum adduction if, for example, the external rectus muscle is cicatricially adherent. The treatment is principally causal.

In paralysis of the facial nerve and the muscles supplied by the oculomotor, trochlearis, and abducens the galvanic current is applied as an adjuvant in several ways.

(1) The entire globe may be galvanized, so that the place corresponding to the paralyzed muscle is invaded by the active, round-plate electrode of 4 to 5 cm. diameter. The indifferent electrode, consisting of a plate 7 cm. wide and 10 cm. long, is, according to Erb and Driver, best applied to the occiput, so that the entire motor tract of nerve conduction may be included. The nape of the neck and the forehead are used for the same purpose. A 1 to 3 Ma. strength current is employed. In all cases care should be taken in making and breaking the current very slowly, applying a very finely graded rheostat, as otherwise unpleasant subjective sensations of light may result and very susceptible children be seized with headache and vertigo.

(2) If the seat of the paralysis is intracranial, galvanization (1- 3 Ma.) transversely through both orbits, from one temporal bone to the other, or from one mastoid process to the other, has been recommended.

(3) Galvanization as in (1) with a small, knob-like electrode pressed as deeply as possible into the paralyzed ocular muscle, the lids being closed.

Each treatment should last from $1\frac{1}{2}$ to 3 minutes. At first one treatment is given daily, later the intervals are longer. They should not produce more than a slight sensation of burning. Improvement is recognized by the degree in which the double images approach each other, and the field of vision, in which only one image is perceived, is enlarged. This may occur rapidly, leading to a cure, or it may extend over several weeks or months. Should galvanization not lead to success after six or eight weeks, it is useless to continue this form of treatment. Simultaneous galvanization of the spinal cord, such as is generally practised in tabes, is said to support the effect by exciting the sympathetic.

Portable apparatus, kept in good condition, can be used, but it must be provided with a rheostat and galvanometer. The electrodes should always be wet and their material in good condition, so as to

prevent caustic effect. The current is best applied during absolute silence, and, after the application is over, perfect rest for ten or fifteen minutes should be enjoined.

Michel recommends "orthopædic" treatment in the following manner: the globe having been anæsthetized by cocaine-adrenalin, the conjunctiva is seized with fixation forceps over the insertion of the paralyzed muscle, and the eye is drawn several times (for one or two minutes) to and fro in the direction of the muscle beyond the border of contraction at either side. Children are apt to offer considerable resistance to this proceeding, but the necessity for resorting to it exists only in cases where early contracture of the antagonist threatens.

As to the value of prismatic treatment, opinions differ. Strong prisms over six degrees are unsuitable, owing to their weight and color dispersion. It is useful to distribute the effect uniformly over both eyes. In paralysis of the external rectus muscle of the right eye, for instance, which can be corrected by a six degree prism, one of three degrees each is placed before each eye, with the bases out. By gradually reducing the strength of the prism, almost fusing the double images, there is no doubt that in prognostically favorable positions increased innervation of the paralyzed muscle will be incited.

In order to prevent accidents, in annoying diplopia, alternate spectacles may be worn. They are provided either with a black metal diaphragm or a transparent glass, so that when the vertigo, occasioned by misorientation, is not excessive, the healthy or the affected eye can be used for vision alternately, and at the same time the paralyzed muscle exercised.

As the paralysis subsides, exercise with both eyes, once or twice daily, is useful, the child attempting to fix with binocular vision a hand which is slowly moved in the motor direction of the paralyzed muscle.

The question of operative measures directed to the relief of diplopia, or of strabismus due to secondary contracture of the antagonist of the paralyzed muscle, should be left to the discretion of the ophthalmologist. This treatment, however, may occasion greater difficulties, under certain circumstances, than removal of non-paralytic strabismus.

A warning should be sounded against premature operative interference, because even after months of stubbornness paralysis may disappear spontaneously, or yield to suitable general treatment.

XVI. DISEASES OF THE ORBIT

1. INFLAMMATIONS OF THE ORBIT

ORBITAL inflammations are very dangerous, both to vision and to life. In addition to tests for leukæmia, pseudo-leukæmia, syphilis and tuberculosis, an examination of the nose and accessory sinuses is indicated. Rhinogenical disease with orbital complications demands, in the first place, early local treatment, while orbital changes due to a general affection are treated according to conditions with arsenic, anti-syphilitic or anti-tuberculous medication. In these cases cure is followed by a prompt retraction of a prolapsed or displaced globe and an improvement of vision.

Affections of the nasal accessory sinuses occur more frequently in childhood, according to Onodi, than has heretofore been supposed. Infectious diseases usually involve the accessory sinuses of children: this is particularly true of scarlet fever. But in the first years of life it is difficult to establish the pathological facts, and, besides, therapeutic measures in that period are rather limited.

In suppuration of the nasal accessory sinuses, the orbital contents become displaced, and are directly affected by the inflammatory process. The optic nerve is especially susceptible, producing a retrobulbar neuritis or thrombosis of the optical vessels. The subjective symptoms are those of neuralgia: pains in the neighborhood of the eyes, especially in the supra-orbital nerve, a sensation of orbital pressure, increased lachrymation and a slight irritation of the conjunctiva, along with other functional disturbances.

The following functional details should be observed in infants and young persons:

Constriction of the orbit and displacement of the globe by dilatation of the accessory sinuses in consequence of a mucocoele; acute, simple, non-ulcerative orbital periostitis, notably after scarlet fever, combined with tenderness of the lower wall of the frontal sinus, with painless muscular paresis (ptosis and paresis of the superior and internal rectus muscles), swelling of the lids, and perhaps protrusion of the globe; and finally subperiosteal abscesses following carious destruction by inflammations adjacent to the orbit,—processes which may also occur simultaneously.

The diagnosis of orbital periostitis secondary to nasal or sinus disease is suggested by the history, the presence of pus, polyps and hypertrophic growths in the nose, tenderness of the orbital roof and inflam-

matory swelling of the lids. If the nasal findings are negative, the onset of paresis of the superior levator and the superior and internal recti, simultaneously with acute coryza, is of importance. In more than fifty per cent. of the cases, the abscesses and inflammations are of rhinogenic origin. They also occur as metastatic processes in endocarditis, pulmonary abscess, pyæmia, etc.; but it is important to note that there are also closed empyemas which cannot be revealed by rhinological examination, and that a primary suppuration of the accessory sinuses may have already healed.

In making a differential diagnosis, it is a noteworthy fact that carionecrosis of the sphenoid may be caused by syphilis and that the only symptom of an empyema of the sphenoid cavity may be an affection of the optic nerve. The early involvement of both optic nerves points to the seat of the primary tuberculous focus in the sphenoid body, as against a posterior ethmoid cell.

In initial optical complication following empyema of the sphenoid cavity, central scotomata are generally due to toxic amblyopia or affections of the optic nerve, consequent upon localized intracerebral affections, etc., while in sinus disease the inflammatory process involves the intracanalicular and intra-orbital parts of the optic nerve first, producing disturbances in the visual field in the form of relative central scotoma for colors. Birch-Hirschfeld, however, observed a central scotoma as an early symptom of sinus diseases, and van der Hoeve an enlargement of the blind spot.

The ophthalmological picture of this neuritis often shows considerable stagnation of the venous flow at the disc, which later becomes a fresh papillitis with blurred borders, radial striation and peripapillary oedema. Choked disc is not often present. In cranial osteomyelitis, which is suggested by inflammatory swelling and abscess formation located outside the territory of the affected cavity, tests for syphilis should be made before operation is resorted to.

Mucocele may be associated with a more or less pronounced protrusion of the globe outward and sometimes downward, and with an elastic, roundish tumor at the upper inner canthus that can be slightly pushed back through a palpable osseous gap toward the frontal cavity of the ethmoid cells. Its beginning is slow and relatively painless. In differential diagnosis must be considered cysts and malignant tumors, including those of the lachrymal glands. However, mucocele may also begin more laterally and behind the supra-orbital notch and be of coarser consistency. Finally, there occur osteophytes, as a consequence of inflammatory irritation in the periosteum of the accessory sinuses, which may simulate exostosis or mixed osteoma. I observed a case of this kind as long ago as 1885.

The *prognosis* of mucocele is good, unless complicated by an acute inflammation. Operation for a small mucocele may lead to a complete return of the normal state, while in large ones, to at least partial correction of the displaced globe. An otherwise uncomplicated, non-ulcerative periostitis may, so far as life is concerned, terminate favorably, but leave behind permanent paresis of the affected ocular muscles. Subperiosteal abscesses may permanently impair the function of the ocular muscles and, when extending posteriorly, injure the optic nerve and involve the brain. After spontaneous evacuation or a simple incision, a fistula usually remains. In phlegmons, especially when accompanied by high fever, permanent operative injury to the optic nerve and transmission to the intracranial cavity is more likely than in ulcerative periostitis. Further details will be found on p. 325. In destruction of the osseous wall of the sphenoid cavity due to syphilis and in deep tuberculosis of the orbit, fatal termination has occurred by the spreading of the cario-tubercular process to the meninges and intracranial cavity. When one considers that even after operations which technically were not particularly difficult fatal meningitis has occurred, it is impossible to make an absolutely favorable prognosis of the major or so-called radical operation on the frontal cavity and ethmoid bone. Nor do the cosmetic results of radical operations from without always give satisfaction; disturbances of the ocular muscles occur often, although they are only transitory, and nasal secretion is not always arrested.

As to the visual disorders, those occasioned by relatively fresh neuritis of the optic nerve are followed by complete restitution of the visual function, unless the papillomacular bundle has been considerably damaged and unless there is absolute central scotoma. However, recurrent closure of the ostium or ostia of the sphenoid cavity may cause a fresh retrobulbar neuritis, even though a broad incision has been made. The orbital section of the optic nerve seems to be particularly endangered by affections of the maxillary cavity. The nerve in the optic canal is equally endangered by suppuration of the accessory sinuses, which are very apt to cause choked disc. The optic nerve may be affected by subsequent intracranial suppuration.

It is an important fact that the acute suppuration of the accessory sinuses in scarlet fever may run such a rapid course that the orbital complication is the first sign of sinusitis.

Treatment in the first place is causal, and particularly rhinologic; the first measures are conservative, although in most cases operative interference will be required later. It is very often possible either to prevent orbital affection or to cure it and non-suppurative orbital inflammations not accompanied by great pain or material elevation of temperature by liberal local application of cocaine-adrenalin and other

remedies, including diaphoresis. In a case of retrobulbar neuritis due to suppuration of the accessory sinuses, slowly but steadily increasing amblyopia was strikingly improved by painting the posterior nares with cocaine-adrenalin during examination.

In fresh, retrobulbar neuritis of uncertain cause, exploratory opening of the ethmoid cells and sphenoid cavity must be considered, unless they are undoubtedly healthy. The surrounding cells may disclose extensive polypoid degeneration, even without noticeable pathological secretion.

In chronic sinusitis and suppuration of the frontal sinus, and in ethmoid cells located well forward, without symptoms of stagnation or fetor, endonasal treatment usually effects evacuation of pus, thereby preventing complications.

Drainage of the affected sinus through the nose is possible when the sphenoid cavity and the cribriform bone are involved, provided the processes are not too fulminant and have not led to perforation. This is true also of mucocoeles of the cribriform bone, whose walls are neither too thick nor too rigid. On the other hand, intranasal operations on suppurating sinuses very often cause an exacerbation of the inflammation, aggravating a previously slight orbital complication so that discoloration of the lids, exophthalmos with fever and tenderness may follow. Even suppurative meningitis with fatal termination has occurred in intranasal evacuation of the ethmoid. The treatment of sphenoid affections with astringent irrigations is not without danger, even in the hands of an experienced rhinologist. After one of these operations a grave orbit inflammation with subsequent panophthalmia followed, and the patient's life was saved only by extensive incision of the orbit from above and outward.

Operative treatment is necessary in mucocoele in non-suppurative orbital inflammation, when the pain increases and the fever rises, and in orbital inflammations accompanied by suppuration or high temperature. In empyema due to transitory occlusion of the ostium, the cavities should be at once incised, unless cocaine-adrenalin causes immediate and permanent opening of the ostium. So far as the sphenoid cavity is concerned, this is nearly always necessary in acute retrobulbar neuritis. In this procedure it is important, especially for the prevention of post-operative meningitis and cranial osteomyelitis, to evacuate the affected cavities thoroughly, and especially an infected suppurative maxillary antrum. Luc-Caldwell's method is the best.

As to operative complications, meningitis has repeatedly been caused by osseous fissures, due to rough operation with blunt chisels. Suppuration of the maxillary antrum is due to caries of the teeth, complicated, perhaps, by suppuration of the frontal cavity and ethmoid cells. Like the latter and pansinusitis, it threatens life itself by subsequent meningitis or cranial osteomyelitis and extradural or cerebral

abscesses. In these conditions, puncture from the medial nasal tract may produce orbital complications, when the lower orbital wall is very near the lateral part of the medial nasal wall or is partly fused with it. Puncture from the lower nasal tract is also dangerous when the orbital base descends deeply into the maxillary cavity. In both cases the orbit can be easily injured, and insufflation of air through the puncture needle may cause emphysema of the orbit; or an attempt at irrigation of the maxilla may cause a suppurative infiltration of the orbital tissue. Detachment of the trochlearis, which is part of Killian's method of operation on the frontal cavity, may lead to permanent disturbance of motility.

2. SEROUS INFILTRATION AND PHLEGMON OF THE ORBIT

Serous infiltration of the orbit originates either from a lid process, as in erysipelas, glanders and anthrax, or from a so-called specific metastasis or pyæmic infection, as in measles, scarlet fever, variola, diphtheria, glanders, typhoid fever, influenza, meningitis or septicæmia. Septic orbital emboli, however, also occur with less striking symptoms.

The affection causes violent pain in the deep parts of the orbit. Cerebral symptoms, such as cephalalgia, vomiting, numbness, slow pulse with more or less elevated temperature and gastric disturbances, are not rare. The motility of the eyeballs is impaired, and the disease displaces the globe, either straightforward or obliquely. The lids are very œdematous and hyperæmic, while the conjunctiva is congested and chemotic. In pronounced cases, the upper eyelid is immovable and drooping. The globe and its vicinity feel hard; should perforation ensue, a yellowish tumor, which later fluctuates, arises at a circumscribed point on the conjunctiva or skin of the lid. With evacuation of the pus all these manifestations are gradually relieved. Inflammation of the optic nerve, compression or thrombosis of its vessels, retinal hemorrhages or detachment, are sometimes complicated by ulceration of the cornea, owing to purulent chorioiditis or defective closure of the lids.

Differential Diagnosis.—Similar symptoms are produced by thrombosis of the cavernous sinus, which has been observed as a metastatic process in measles and scarlet fever. Its occurrence is often due to caries of the petrous bone, to dental suppuration or phlegmonous tonsillitis, or the result of septic thrombosis of the orbital veins secondary to the orbital inflammation,—as, for instance, after chronic erysipelas of the lids. In sinus thrombosis, however, there are doughy œdemata in the mastoid region.

In marantic thrombosis, which generally attacks the superficial sinuses, ocular symptoms are relatively rare, certainly rarer than in the phlebitic form. Thrombosis resulting from inflammatory processes in the orbital region usually affects the sinus cavernosus.

It is distinguished from metastatic purulent ophthalmitis, in which the infiltration always involves the adjacent orbital fat more or less, by the fact that the ocular contents are not diseased. Secondary inflammatory changes in the ocular fundus are entirely absent in non-purulent tenonitis, but they have been observed in influenza and erysipelas. Perforation of the globe does not occur. A circumscribed orbital infiltration, which occurred in a few cases of influenza with severe supra-orbital neuralgia, underwent complete involution when treated with hot applications.

The earlier the abscess is drained the better is the prognosis in metastatic orbital inflammations, as suppurative meningitis and cerebral abscess are thus prevented. Nevertheless, atrophy of the optic nerve may reduce vision decidedly. It may indeed be completely destroyed by detachment of the retina or vitreous infection, or by panophthalmitis following suppurative corneal infiltrates.

Metastatic bilateral orbital phlegmon, which is a very rare affection, has been observed as a suppurative thrombosis of the sinus cavernosus and the orbital veins in measles and other affections. Its prognosis is extremely unfavorable.

Treatment.—Ice compresses will often relieve pain. The most important point, however, is to prevent the spreading of the suppuration toward the cranial cavity. With this end in view, the retrobulbar pus focus is opened, as early as possible, by a deep puncture with a pointed scalpel at the place where the suppuration is suspected, or where the fluctuation is most distinctly felt. The wound is enlarged after the primary drainage. The cavity is then carefully irrigated with a 3 per cent. boric acid solution, loosely packed with iodoform gauze, and covered with a monocular bandage. This is repeated daily, until the abscess has completely healed. Should there be loss of corneal substance from a purulent process, the bandage should be a duplex one, as it accelerates healing. Simultaneous purulent chorioiditis requires treatment as described on p. 253. Cases of this kind should, therefore, be referred to the ophthalmologist without delay.

Non-suppurative tenonitis undergoes the quickest involution under a pressure bandage. Diaphoretic treatment may likewise have a favorable effect.

3. ORBITAL PERIOSTITIS

This affection has been observed in variola with caries of the orbital margin. It is characterized by local tenderness, elevation of the osseous border at some point on the orbital rim and by œdema of the lids. When swelling of the orbital margin is very pronounced, the globe may be displaced toward the opposite side of the pathological focus.

As distinguished from orbital phlegmon, which may also occur, together with periostitis, it is to be noted that in phlegmon the eye is usually displaced forward. The lid in orbital phlegmon is generally blue-red, in periostitis pale red. It may sometimes be necessary, however, to make an incision and inspect the affected bone in order to establish the diagnosis. This incision requires great care, on account of the danger of perforating the orbital bone, which, owing to the very thin orbital roof, is a matter of considerable moment.

In periostitis situated in the deeper parts, the differentiation of retrobulbar phlegmon is impossible, until outward perforation of the periosteal abscess has taken place.

Treatment.—In order to prevent meningitis and cerebral abscess moist heat should be applied, to hasten involution. If pus forms (fluctuation), the same measures are required as in metastatic orbital phlegmon. The shedding of particles of carious bone may demand surgical treatment; so will lid adhesion to the bone caused by the defect of the osseous orbital border, especially if these adhesions lead to shortening and disfigurement of the lids (ectropion, etc.).

Syphilitic orbital periostitis is often seen as a chronic periosteal thickening, very often beginning in the orbital roof. It is rarely congenital, but is not infrequently a late manifestation in acquired syphilis. Hyperplastic periostitis in the depth of the orbit, where again the upper wall is commonly involved, may cause protrusion of the globe and pressure on the optic nerve. Here again the palpebral swelling is usually less than in affections of the orbital border. Protrusion of the globe, compression and inflammation of the optic nerve, paralysis of several or all the ocular muscles and of the trigeminus, point to involvement of the superior orbital fissure.

Symmetrical bilateral involvement, periostitis at any part of the body, either present or healed, spontaneous pain at night, point to syphilitic periostitis of the orbit.

Treatment.—Internal use of large doses of mercury and potassium iodide—or possibly of salvarsan—may cause the syphilitic periostitis thickening and the retrobulbar infiltrations to disappear in a month or two, leaving at most a few roughened areas on the bone. Repeated painting of the upper orbital margin with tincture of iodine has a favorable effect in the initial stage of periostitis. Drainage of the focus is necessary in exceptional cases only.

4. TUBERCULOSIS OF THE ORBIT

This affection is by no means rare. Tubercular periostitis and ostitis of the orbital wall is a relatively benign affection, always occurring in the anterior part of the orbit. The disease may also be caused

by a comparatively slight trauma, such as contusion. It does not, as a rule, spread to the orbital tissue, globe or optic nerve. Tubercular disease of the orbital ostium starts from the periosteum of the orbital walls, the lachrymal glands or sac, or from the chorioid. Deep tubercular processes are due either to a spreading to the frontal cavities and orbital roofs, or to primary tuberculosis of the sphenoid body, the sphenoidal sinus and the posterior ethmoid cells. The fact that the nose and accessory sinuses may appear clinically unchanged is explained in several ways: The infantile nose is very narrow, and posterior rhinoscopy is impossible, so that with but a slight swelling of the turbinates it is impossible to inspect the deep parts. Illumination of the accessory sinuses is equally unsatisfactory. It is still to be determined whether and in

FIG. 41.



Leukæmic tumor of the orbit (lymphosarcomatosis).

how far the orbit is involved in primary tuberculosis or secondary tubercular inflammation of the maxillary cavity, such as may spread from the alveolar process or the oral cavity, in consequence of tooth extractions or lesions of the gums.

Retrobulbar tuberculosis of the middle and posterior part of the optic nerve should also be considered, as it is generally associated with a tubercular affection of the base of the skull, and especially of the meninges.

Contrary to the stormy inflammatory manifestations which acute suppuration of the ethmoid cells causes in the orbit, orbital changes in grave tubercular affec-

tions at the apex of the orbit set in late. On the other hand, the exceedingly early involvement of the canalicular portion of the optic nerve, due to compression of tuberculous granulations spreading from the sphenoid, is an important diagnostic fact. It is recognized with the ophthalmoscope as a pressure atrophy, or a slight inflammatory change at the optic disc. The visual disturbances vary, according to whether there is a simple compression of the optic nerve or direct inflammation. Displacement of the globe, exophthalmos and the involvement of the other orbital structures are milder. According to Lehnhart, disorders of the ocular muscles are absent, because in affections of the sphenoid body the optic nerve is involved, but not the nerve trunks passing through the superior fissure.

Tubercular periostitis and ostitis in the anterior section of the orbit is decidedly more frequent. These processes lead to circumscribed supuration and fistulæ of the skin covering the lids and rarely involve the orbital tissue, globe or optic nerve.

Treatment.—Early and radical removal of the superficial or moderately deep tubercular foci by means of a curved incision in the superciliary ridge. If necessary, the incision should be continued down to the nose until the bone is reached, the periosteum should be elevated and the caseous masses removed by thorough, though gentle, scraping of the infected area. Affected foci at a somewhat greater depth may have to be destroyed by careful thermocauterization of the diseased cavities and their walls. This should be followed by packing the open wound with gauze. But recently I cured a nine-year-old boy in whom tuberculosis had invaded the upper nasal part of the orbit from the left anterior ethmoid cell. If the processes are located deeply in the orbit, exposure of the carious sphenoid and optic canal by eversion of the nose must be considered.

In a case of exenteration of the orbit described by Lebenhart, an eye, which had unquestionably been blind for a long time, had to be sacrificed.

5. ORBITAL HEMORRHAGES

Subperiosteal hemorrhages in Barlow's disease, when occurring in the orbital roof, manifest themselves by exophthalmos which is usually accompanied by superficial hemorrhagic discoloration of the lids, and terminates sharply at the upper orbital border. Isolated orbital hæmatoma with exophthalmos is rare. Like palpebral hemorrhages, it is often caused by violent crying. Occasional orbital hemorrhages occur also in hemorrhagic diathesis, especially in hæmophilia. They are seldom spontaneous, but are the result of injuries.

The *treatment* is causal or locally symptomatic.

6. NEOPLASMS OF THE ORBIT

Tumors of the lymphatic tissue occupy the foremost place among new growths of the orbit. According to Meller, they start from the interstitial connective tissue of the lachrymal glands and the conjunctival mucosa. In the absence of ducts in the orbital tissue for the escape at cells, they must remain in the orbit. Consequently displacement of the globe supervenes, and it may finally be completely encircled by the new growth. Blood examinations have shown that these infiltrations, in which Meller includes chloromata, are a local phase of leukæmia, pseudoleukæmia and lymphomatosis which terminated fatally as shown in Fig. 41.

The tissue proliferations of lymphatic leukæmia, which are either unilateral or bilateral, soon become apparent even to the layman, be-

cause of early exophthalmos, immobility of the lids, and diplopia. These symptoms are all a consequence of leukæmic neoplasms which have in part invaded the lachrymal glands and retrobulbar tissue.

There may also be a question of lymphoid tumor, originating by ligation and proliferation of conjunctival follicles into the orbit.

In the further course of the diseases there may be keratitis and lagophthalmos.

Treatment.—Continued use of arsenic in large doses has repeatedly proved successful in orbital lymphoma. X-ray treatment has also been successful.

Atrophy of the orbital fat tissue and subsequent ptosis of the lids, and sunken eyeballs (enophthalmos) are prominent signs of marantic conditions, such as manifest themselves in Asiatic cholera, vomiting and purging, and as a sequel to other infectious diseases.

As the general condition improves, the globes resume their normal positions.

In contradistinction, the reduction of the orbit and palpebral space in hemiplegic infantile cerebral paralysis is unilateral. So is enophthalmos, which is generally the result of long-standing paralysis of the cervical sympathetic. It is also characterized by narrowness of the palpebral fissures, occasioned by paralysis of the smooth muscle fibres of the upper lid, by a reduction of intra-ocular tension, and by contraction of the pupil. The vessels of the paralyzed side of the face are usually dilated at first, but later constricted. Accordingly, the affected half of the face is at first redder and warmer, later paler, or there is hyperhidrosis and hydrosis. In most cases there is also emaciation of the affected half of the face. There are no functional disorders of the eye that are noteworthy. Most of these cases are not susceptible of cure.

XVII. INJURIES OF THE EYE

ALL serious ocular injuries belong to the domain of the ophthalmic surgeon. First aid is of great importance. It should consist in cleansing the traumatic area with a slightly warmed, freshly prepared 3 per cent. solution of boric acid or sublimate 1 to 5000. The surgeon's hands should be thoroughly scrubbed and all instruments which come in contact with the injured eye and the neighboring tissues carefully sterilized. Rough handling must be avoided, especially in injuries which have cut the membranes of the globe. In these cases nothing should be done in the way of first aid but to apply a well-fitting bandage, which should preferably be a duplex one. The next step is to put the patient in the hands of an ophthalmologist by the quickest available means. This applies particularly to traumatic subconjunctival ruptures of the sclera, as only very early appropriate treatment can achieve favorable results.

Gastro-intestinal auto-intoxication may be prevented by internal antisepsis (inunction, sodium salicylate, aspirin, etc.), or by a diet composed largely of milk and vegetables, with very little meat, and by careful regulation of the intestinal function, especially in children of low general tone and those with congenitally abnormal eyes. Even in an apparently favorable course, the healthy eye should be constantly controlled by the uranin test (p. 234). This is especially important when a supervening uveitis does not improve after a few weeks. It is also an unfavorable sign if the intra-ocular pressure decreases, or if retinal detachment or phthisis of the globe with retraction of the scar should follow.

In general the prognosis in ocular injuries is less favorable if complications with acute infectious diseases develop. I observed, for instance, a cut wound in the eye of a ten-year-old girl which progressed favorably until the onset of scarlet fever caused an exacerbation which led to panophthalmitis. The same sequence was observed in a sixteen-months-old baby who had scalded an eye.

Medico-legal opinions in difficult cases should be given by an ophthalmologist.

It is important to note that in traumatic loss of substance and in ulcers of the cornea, vascularization is absent, especially when the wound does not become infected.

1. FOREIGN BODIES IN THE CONJUNCTIVA, LACHRYMAL PASSAGES AND CORNEA

Sand, powder or very small particles of stone are often lodged in large numbers in the conjunctiva and cornea, and may be so deeply imbedded that they must be left until loosened. To relieve violent pain,

however, a little cocaine ointment (0.2 cocaine, 0.002 sublimate and 10.0 vaseline) may be introduced into the conjunctival sac, and a moist bandage (cataplasm Langlebert or acetate of aluminum) applied. Powder grains in the lids are removed later by galvanocautery with a fine, weakly red-glowing point.

Isolated foreign bodies in the conjunctiva, when located in the flat sulcus over the fornix of the upper lid (sulcus subtarsalis), caused considerable pain and irritation because with every movement of the lid the foreign bodies rub upon the globe, and especially upon the cornea.

They are removed by everting the upper lid with the aid of a glass rod and wiping them off with a sterilized cotton tip. In the same way foreign bodies are removed from the region of the internal canthus or the lower fornix.

Foreign bodies hidden behind the lower lid or in the superior fornix may be of considerable size, such as a splinter of wood, a barb of corn, hay or straw, or so-called astacoliths. At first they cause but little annoyance, if so located that they do not move during lid movements and the eye is not rubbed with the finger. Foreign bodies located on the conjunctiva of the lower lid can be easily removed by drawing away the lower lid and wiping towards the nose across flattened conjunctival sac. Foreign bodies located in the upper fornix frequently do not cause irritation at first. In order to locate them, cocaine is instilled and the upper lid is everted. While the patient looks downward, the right thumb is placed flat upon that portion of the globe covered by the lower lid, and the globe is pressed gently but firmly backward. The everted lid is then drawn slightly upward with the left thumb, the child still looking downward, when the upper fornix will protrude like a ridge, and admit of an inspection of its entire extent. By gently moving the upper lid downward, the foreign bodies will be made to escape from the upper fornix, where they were hidden. If the child cannot be induced to look downward, the fornix must be pressed downward with a sound applied along and above the upper lid. This procedure, too, can be facilitated by the application of cocaine.

Should the foreign body be allowed to remain firmly adherent in the retrotarsal fold for any length of time, polypous formation will take place around it, so that it can be found only with difficulty, except with the aid of a Daviel spoon. If this instrument proves ineffective the child is placed in the recumbent position and instructed to look downward. If this instruction is not obeyed, the globe must be turned downward with sterilized forceps. The convex cartilaginous border of the everted upper lid is then turned up with a spatula and the foreign body dislodged from the granulations with a Daviel spoon or forceps. The granulations need not be removed unless that is necessary for the removal of all parts of the foreign body.

In infants too young to give any useful information polypous proliferations in the conjunctiva should arouse the suspicion of a foreign body.

Foreign bodies in the bulbar conjunctiva need be removed only if they give rise to irritation. As a rule, however, they protrude beyond the conjunctival surface in the shape of a spike or eminence; a similar effect is sometimes produced by the lime infarcts of the Meibomian glands, which act like foreign bodies. They are removed with a cataract needle or small forceps, the bulbar conjunctiva being incised, if necessary, with a Graefe cataract knife (Plate XXI, Fig. 3); a bandage is applied until the wound has healed. It may be necessary to dissect

FIG. 42.



Eye pad.

away a small piece of the bulbar conjunctiva. To relieve pain following the removal of foreign bodies from the conjunctiva or cornea, cold compresses are applied. Similar treatment is indicated, should there be hyperæmia or œdema of the conjunctiva. The latter condition is usually confined to a circumscribed area in the tarsal conjunctiva and in the fornix of the inferior conjunctiva, and may be associated with pronounced hyperæmia and chemosis of the bulbar conjunctiva. This is an important point for differential diagnosis. To relieve severe pains, $\frac{1}{2}$ -1 per cent. cocaine ointment may be inserted into the conjunctival sac, and an eye pad worn until the irritation subsides (Fig. 42).

Excessive lachrymation and severe conjunctival irritation may also be caused by foreign bodies in one of the lachrymal puncta or canaliculi. Cilia, bits of hair, bristles, wood, seeds and husks have been found in them. They are best removed with ciliary forceps (Plate XXI, Fig. 8), using a loupe, if necessary. Smaller particles, and those located more deeply,—such as iron, lime, stones or concretions of fungous masses—are removed, with a fine cataract needle, after instillation of cocaine, great care being taken not to injure the canaliculus.

In one case I removed a fungous concretion and saved the canaliculus by gradual dilatation of the lower part by the conical sound and syringing the upper part with a sublimate glycerine solution (sublimate 1.0 : 10.000 in aq. dest., glyc. 5ā) under gradually increasing piston pressure. The waste water escaping by the lower canaliculus loosened the concretion, so that by massage upon the skin over the canaliculus and conjunctiva with a Daviel spoon the entire contents were evacuated through the lower punctum. The syringing procedure having been repeated, I very carefully cauterized the mucosa of the fundus of the cavity with a little silver nitrate fused on the point of a conical sound. This led to complete restitution of normal lachrymal secretion.

Should this procedure be unsuccessful, the canaliculus must be slit with Weber's knobbed lachrymal knife.

Should there be a tendency to perforation outward, an incision is required parallel to the palpebral border. If there is a likelihood of spreading to the lachrymal canal, the lachrymal sac must be incised.

Foreign bodies in the cornea are usually located in the area corresponding to the palpebral fissure. Their removal depends upon whether they are recent or have been imbedded for several days; and whether an attempt to remove them has already been made. They are most easily found by letting light from a window fall on the eye, the latter being moved in all directions. This will show light foreign bodies upon the background of the pupil, while the dark ones may be seen upon the lighter background of the iris. This method will also show the reflex pictures of the window interrupted by the foreign body. In doubtful cases fluorescein may be instilled. Particles of iron are generally oxidized, and therefore have a dark brown or black appearance (ferrous oxide). If they enter while aglow, they will be first surrounded by a white ring which soon changes to a red-brown fringe—the so-called rust-ring, which is formed by a solution of iron and a precipitate of ferric hydroxide. Minute specks, due to an explosion, often heal without causing reaction. In other cases there may be a reactive corneal supuration, and in order to prevent such a contingency, the foreign body should be removed at the earliest possible moment. Those located superficially are removed as follows: Local anæsthesia with several

PLATE XL



FIG. 1.
Bismuthus
hook.



FIG. 2.
Bismuthus
hook.



FIG. 3.
Bismuthus
hook.



FIG. 4.
Bismuthus
hook.



FIG. 5.
Bismuthus
hook.



FIG. 6.
Bowman's
sound.



FIG. 7.
Lachrymator.



FIG. 8.
Lachrymator.



FIG. 9.
Lachrymator.

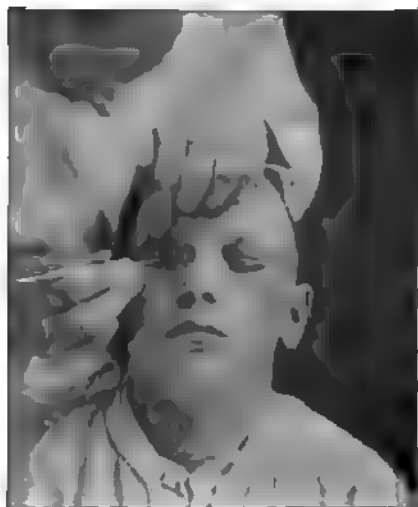


FIG. 10.
Gold and silver
syringe.

7

drops of 2 per cent. cocaine, to which may be added a few drops of suprarenin (1.0 : 1000.0). Keep the lids apart with the left hand; wipe or gently rub off the foreign body with a firmly twisted, sterile cotton tip. If this is unsuccessful, and for particles lying at greater depth, remove with a clean, well-made foreign-body needle (Plate XXI, Fig. 2) (Fig. 43). The child sits on a chair facing a bright window, while the physician stands behind, rests the child's head against his chest and holds the lids apart with the fingers of one hand, using gentle pressure in such a way that the eye receives firmer support. The patient is instructed to look in that direction which best brings the foreign body into view. The foreign body is now immediately approached from behind with the point of the needle. The foreign body is dislodged

FIG. 43.



Removal of a foreign body from the cornea.

FIG. 44.



Monocular bandage.

without penetrating deeper than is absolutely necessary. In order to prevent accidental injury to other parts, especially unnecessary damage to the epithelium, the spud is held very lightly, and its movements should always be forward (Fig. 43). Particles of a rust-ring are removed most easily by handling the point of the needle in the way a rubber eraser is used. Finally, a little cocaine or boric ointment should be instilled in the conjunctival sac and a monocular bandage applied (Fig. 44) with directions not to touch the eye for a day or two. If the foreign body is surrounded by a gray, infectious ring with blurred borders, and if there is a deep pericorneal injection, or if the iris is inflamed, the site of the foreign body after removal should be carefully touched with tincture of iodine. A moist bandage (1 per cent. aluminum acetate) is then applied, to be changed once or twice a day, until the corneal defect

has healed. Superficial abrasions of the cornea can be made clearly visible by instilling fluorescein. Such abrasions may cause great pain, but it is rapidly relieved by a borated sublimate or iodoform ointment compress, and instillation of a 5 per cent. solution of dionin or aristol oil. Cocaine is to be avoided, owing to its injurious effect upon the corneal epithelium. Healing may be retarded by renewed excoriations, occurring weeks later at the original place of injury.

Injuries with a copying pencil have varying effects. The blue discoloration may disappear in a single day, but as the methyl-violet used in its manufacture is an inflammatory agent, there may also be conjunctival irritation, accompanied by considerable photophobia, croupous conjunctivitis, ulcers and ulcer of the cornea. In all cases, therefore, it is advisable to remove the particles from the conjunctiva as well as from the cornea.

The removal of foreign bodies from the cornea is facilitated by an illuminating apparatus. A Sidler-Huguenin's lamp is placed at the side of the patient, while a lens, fastened to the forehead of the patient with a rubber band, is so concentrated upon the cornea that the physician, sitting in front and using a magnifying glass, can recognize the foreign body with great distinctness. Kehr's model is simpler (Fig. 29, p. 195).

A steady hand is a requisite, particularly in treating restless and excited children, or the cornea may be perforated. Children with a foreign body deep in the cornea or protruding into the anterior chamber should therefore, after application of a temporary protective bandage, be referred to an ophthalmologist without delay. The removal of a foreign body that has penetrated the cornea is easier when its anterior end protrudes through the cornea, so that it may be grasped and extracted.

In this way, using a coagulum forceps, I once removed a very thin bee's sting about 2.5–3 mm. long, which had penetrated the cornea about $1\frac{1}{2}$ mm. away from the lower corneal margin in such a way that, running obliquely upward and inward through the anterior chamber, its point stuck in the iris.

Powder, minute particles of stone or lime, even in large numbers, when embedded in the cornea, often heal without causing reaction. They are therefore best left alone, in order to avoid ulcerous scars. As a rule, cold compresses or a monocular bandage as a unilateral occlusion of the eye are sufficient.

2. OPHTHALMIA DUE TO CATERPILLAR HAIRS

(OPHTHALMIA PSEUDO-TUBERCULOSA OR NODOSA)

This affection is caused by hairs of caterpillars. It begins with œdematous swelling of the lids, redness of the palpebral skin (similar to that produced by superficial corrosion or burning of the eye), blepharo-

spasm, photophobia, lachrymation, violent, piercing and burning pain. The superficial and deep circumcorneal congestion is usually accompanied by retiform, finely striated opacities in the inner or outer lower quadrant of the cornea with light stippling of surface, and erosions in which more or less numerous yellowish, brownish, light ash-gray or greenish little hairs can be seen, in a few days or weeks. Around and between them are epithelial and subepithelial limpid vesicles, leaving numerous small, dense, punctiform or striated infiltrates or spots. These may disappear in a month or two, if the caterpillar hairs shrink without penetrating deeper, so that the cornea again becomes transparent with complete or nearly complete restoration of vision.

If the conjunctiva contains hairs, they are usually found in the dependent portions of the palpebral and bulbar conjunctiva and the inferior subtarsal sulcus, less often in the superior tarsal conjunctiva. Depending upon location, their presence occasions swelling of the palpebral or bulbar conjunctiva.

It is important to remember that the disease is one which occurs only during the late summer and fall, when certain kinds of caterpillars appear in great numbers.

Unless the hairs are removed promptly, miliary, gelatinous, semi-transparent nodules appear from time to time in groups of varying numbers. With every new group the eye becomes injected. In the tarsal conjunctiva and fornix the eruptions have a reddish-gray appearance. Those in the bulbar conjunctiva at first appear reddish-yellowish, later yellowish-gray, and may be either movable or firmly adherent to the sclera. These nodules contain fine hairs which can be seen with the loupe. They stand partly oblique, partly erect on the surface of the globe, while others protrude a short distance. In cases which have been treated correctly from the first, the nodules may gradually become smaller and disappear in the course of one to five weeks without removal of the hairs, while the slight ciliary injection and mild hyperæmia of the iris correspondingly decrease.

In patients treated in advanced stages, the visual function gradually declined with intermittent exacerbations and plastic iridocyclitis, which may extend over six months and more, and in one case over two and one-half years.

In those cases the nodules and hairs are generally found in the lower half of the conjunctiva, episclera and cornea. The cornea becomes vascular, densely infiltrated with striated spots and sometimes swollen, while in old cases it becomes opaque through scar formation. In a few cases there was almost absolute pupillary occlusion and seclusion, nodules in the iris, which here again were most marked in the lower half of the membrane. Radial folds and cicatricial bands are sometimes seen

The first stage of the disease is characterized by a redness of the eye, which is accompanied by a discharge of tears. This is followed by a swelling of the eyelids, and the eye becomes very painful. The patient is unable to open the eye, and the vision is greatly impaired. The disease is caused by a virus, and is highly contagious. It is most common in children, but can also affect adults. The disease is usually self-limiting, and the symptoms will resolve themselves within a few days. However, in some cases, the disease can lead to more serious complications, such as blindness. It is important to seek medical attention if you suspect you or your child has this disease.

The second stage of the disease is characterized by a more severe inflammation of the eye. The redness and swelling are more pronounced, and the discharge of tears is more profuse. The patient is now unable to see, and the eye is in great pain. The disease is now in its most severe stage, and the patient is in danger of losing their vision. It is important to seek medical attention immediately at this stage. The doctor will prescribe strong anti-inflammatory drugs to reduce the inflammation and pain. In some cases, surgery may be required to remove the infected tissue. The disease is still highly contagious, and the patient should be isolated from others to prevent the spread of the virus. The prognosis is now very poor, and the patient's vision may be permanently lost.

The third stage of the disease is characterized by a more gradual recovery. The inflammation is subsiding, and the patient is able to see again. However, the vision is still very poor, and the eye is still sore. The disease is now in its final stage, and the patient is recovering. The prognosis is now good, and the patient's vision should return to normal within a few weeks. It is important to continue to follow the doctor's instructions to ensure a full recovery.

retain their irritating, caustic effect for a long time—three years, according to Gossens.

Treatment.—In very mild cases, cold compresses should be immediately applied for such periods and at such intervals as the violence of the subjective and local manifestations indicate. In severe cases, the patient should be immediately referred to an ophthalmologist. Repeated touching of the affected parts of the cornea with absolute alcohol may be used to absorb the formic acid contained in the hairs, and to prevent reaction from this source.

It is not improbable that the migration of the caterpillar hairs to the deep parts is effected by their perforating the posterior layer of the cornea, and in this way gaining access to the iris, such migration being assisted by the violent blepharospasm and rubbing. The first thing should therefore be to remove the visible hairs,—those in the conjunctiva with very fine forceps, those in the cornea with a foreign-body needle, in each case using a loupe if necessary. The subjective manifestations are alleviated by instillation of atropine and cocaine, two or three times daily. The application of an occlusion bandage and moist, warm compresses has been very effective in a few neglected mild cases. Inunctions with mercury ointment and leeches have several times been used to advantage. Excision of the nodules in the conjunctiva, and removal of the hairs embedded in the conjunctiva by squeezing between the blades of fine forceps, have been successful. These procedures may be facilitated in some cases by the fact that the conjunctiva is tightly drawn over the nodules, and that the resiliency of the stiff hairs tends to bring them to the surface. The hair-like structures in the corneal infiltrates are dislodged with a foreign-body needle, great care being taken to injure the tissue as little as possible. When there are hairs in the iris also, the area involved should be removed by iridectomy so as to prevent further iritic irritation.

Conjunctival nodules, resembling trachomatous follicles, which had formed around invading plant hairs, probably cynarrhodon, were observed by Schmidt-Rempler and Marcus.

3. FOREIGN BODIES IN THE INTERIOR OF THE EYE

Generally speaking, it is more difficult to demonstrate a foreign body in the interior of the eye. This is particularly the case when the wound in the sclera or cornea is very small and closes rapidly, when minute splinters are fastened in the ciliary body and cannot be seen, even though the pupil is considerably dilated, or when the injury is old. It may also happen that, owing to traumatic cataract or fibrinous exudation into the internal eye, lateral illumination and ophthalmoscopic examination are no longer possible, or that the foreign body is hidden

behind the iris or at the ciliary body. In some cases it perforates the posterior bulbar wall, or is retained in the orbit. Rapid removal of the foreign body is often urgent, so that the patient should be referred immediately to an ophthalmologist.

Even sterile foreign bodies which have penetrated the globe may many years later produce an iridochorioiditis, which will necessitate enucleation of the globe.

Copper, for instance, almost immediately produces either purulent iridocyclitis or vitreous abscess, rapidly destroying the eye. Even though the inflammation may be happily overcome, chronic iridocyclitis or retinal detachment often follows in its wake. The dissolution of minute particles of iron splinters may, years afterward, cause corrosion of the eye, notably the lens, iris, ciliary body, retina and optic nerve (siderosis bulbi), accompanied by hemeralopia, concentric restriction of the visual field and reduction of the central visual acuity. The iris, especially at the pupillary margin and its immediate neighborhood, assumes a strikingly yellowish-green and later a chestnut-brown, tinder-like appearance. At the inner surface of the anterior part of the lenticular capsule, even though the splinter is not within the lens, a precisely semicircular wreath of rust-brown spots becomes visible, formed by epithelial proliferations with precipitates of ferric oxide which resemble ruptured posterior synechiæ still adherent to the capsule. As the lens loses its transparency, it becomes a diffuse yellow and later brown.

Vitreous opacity and shrinking, painful iridocyclitic irritations, accompanied by frequently recurring hemorrhages and deposits of ferric oxide salts on the posterior surface of the cornea, will necessitate enucleation, unless the iron splinter can be removed. A similar impregnation may be present at the latest stage, though rarely in the conjunctiva and sclera. It is an exception when an eye remains permanently free from subjective reactions. In dissecting an eye which had been enucleated on account of secondary glaucoma due to dislocation of the lens, I once found a large iron splinter in the ciliary body, so firmly encapsuled that it was dislodged only with the greatest difficulty. The patient then remembered an injury which had happened to his eye twenty years before.

The *prognosis* of foreign bodies in the interior of the eye is always a very serious one, but, nevertheless, there are fortuitous results. Friedenwald, for instance, observed a cure with full visual acuity in two cases of double perforation of the globe, one caused by copper splinters and the other by bird shot. In both cases the sclera was perforated near the limbus, as were the ciliary body and the posterior chamber. The foreign bodies left the globe through the sclera near the limbus and were found under the conjunctiva. In neither case was there an injury to the lens, merely iridodialysis remaining.

Intra-ocular and orbital foreign bodies are revealed by illuminating the sclera with reflected light (comp. p. 20), or their presence is revealed by pain upon application of the magnet, and by sideroscopy with the iron finder of Asmus or Koster. Unfortunately, the latter method furnishes no information as to whether the splinter is inside or outside the globe, and splinters which have healed subcutaneously within the vicinity of the eye also give a positive reaction.

Nor does the X-ray always locate them exactly. A double shadow, for instance, on a plate taken by Koehler's method, does not show positively that the foreign body is located in the globe, because, if located behind the eye, it does not always remain immovable during the movements of the eye; on the other hand, a single shadow points to extra-ocular location or a double perforation of the eye. Nor can the exact seat of the foreign body be always definitely located by the methods of Fränkel, Holth and Sweet.

Wessely determines the seat of intra-ocular foreign bodies with the X-ray by inserting a thin, shell-like prothesis into the conjunctival sac. The part corresponding to the cornea consists of glass highly impregnated with lead, so that this part appears as a very dark shadow, the rest of the prothesis as a lighter one. "In this way, the position of the globe can be clearly determined in the X-ray pictures, and frontal as well as lateral pictures, taken in various directions of vision, allow the exact determination of the seat of the foreign body."

The X-ray procedure described by Gillet for localizing foreign bodies which do not consist of iron may be useful for iron splinters which cannot be localized by ophthalmoscopic sideroscopic examination, or by the electromagnet.

Treatment.—An iron splinter, which has just penetrated the sclera or interior of the eye, should be extracted by the electromagnet as soon as possible. Particles located in the scleral layers are sometimes obscured by a subconjunctival hemorrhage. The diagnosis of a foreign body is made by the bulging of the overlying conjunctiva which takes place when the magnet is applied.

If an iron splinter in the lens is removed through the still patent or operative wound, the transparency of the lens may be preserved.

According to my experience, an important point in treatment is the frequent instillation of mydriatics to maintain effective tension of the iris, and the wearing of a duplex bandage for at least a week, in order to exclude completely accommodative changes in the lens.

The gentlest way of removing the iron splinter from the internal eye is to propel it around the lens through the pupil into the anterior chamber, whence it is extracted, with Hirschberg's hand magnet, through the original perforation or a new incision. Lateral removal of the splinter

through a meridian incision in the sclera and introduction of the hand magnet into the vitreous, as is practised in initial or developed siderosis, is not devoid of danger. It has very often led to subsequent retinal detachment. If an iron splinter, that has lodged in the globe for a long time, shows no disposition to create trouble, there is no need to remove it. The extraction of an iron splinter through the pupil with a strong magnet is done with the patient in the sitting posture, so as to prevent the sudden drawing back of the head. Should the splinter be located in the posterior part of the eye, it will often have to be sidetracked first toward the equator, after which it can be drawn forward. Care should always be taken that it does not become entangled in the ciliary body or iris, or lodge where it is not wanted. The magnets used are the giant magnet of Haab and the inner-pole magnet of Mellingner-Haller. The latter "combines the advantages of the small handy Hirschberg magnet with those of the giant magnet" (A. Vogt).

Should infectious germs enter the eye with the foreign body, the latter must always be removed in order to prevent or alleviate infectious inflammation. Removal of percussion caps or copper splinters from the globe is rarely successful. With numerous foreign bodies in the interior of the eye from powder or dynamite explosions, this is nearly always impossible; particles in the cornea usually heal without reaction.

With lead, there is little, if any, chemical action. Small shot heals by encapsulation, provided it is entered sterile. Even shot which has perforated the posterior wall of the globe often remains in the orbit or vitreous without reaction; this is true also of revolver bullets and similar large projectiles. Glass, wood, porcelain and stone, if sterile upon entrance, behave in the same way. Yet a pearl tumor of the iris has often been caused by a ciliary hair. It should, therefore, be removed, as the permanent removal of the neoplasm is only rarely successful. There are constant recrudescences which, by subsequent glaucoma, may even render enucleation of the globe necessary.

Migration of an intra-ocular foreign body—from the vitreous into the anterior chamber, etc.—has often been observed.

In view of all these points, patients with foreign bodies in the anterior chamber or depth of the eye should be referred immediately to an ophthalmologist. Careful and thorough cleansing of the lids and conjunctival sac, together with an aseptic first aid bandage, is all that should be done in the way of preliminaries.

Here and there an intra-ocular foreign body may be successfully removed without its leaving visible trace. I succeeded in one case in removing a piece of brick the size of a pinhead from the lower temporal quadrant of the iris.

The fact that every injury of the eye due to foreign bodies deserves the most careful attention is proved by the cases which develop retro-

bulbar abscess and tetanus, the latter especially after the entrance of wood splinters. In order to remove larger, impacted foreign bodies the traumatic canal may have to be amplified, in order to evacuate any accumulation of pus or prevent its formation.

Disturbance of the ocular muscles in tetanus infection due to injury of the eye and its adnexa is shown not only by the *facies tetanica*, but also by the uniform, bilateral, exceedingly small palpebral fissure due to permanent contracture of the sphincter. There is spastic contracture of the superior levator muscle of the lid and rigidity of the corrugator supercilii, and, besides, when the tetanus toxin comes into direct contact with a nerve of the ocular muscle, there will be continuous spasm of all the muscles of that eye, which, after a short time, radiate to the other eye and the muscular region of the facial nerve, etc.

Local tetanic rigidity of the pupil may also ensue. On the other hand, in tetanus descendens, which is nearly always accompanied by trismus, the muscles of the oculomotor nerve, unlike those of the lid, do not seem to be involved (*Salus*).

4. EXTERNAL INJURIES OF THE EYE

The prognosis of injuries to the lid corruptions by lime, acids or alkalies, etc., is not unfavorable at the onset, unless the conjunctiva and cornea are involved. In many cases only the superficial layers suffer, but by subsequent shortening of the palpebral skin complications may ensue, such as ectropion, ptosis and, if the palpebral margin is involved, entropion, trichiasis, symblepharon, corneoblepharon and ankyloblepharon. The same result follows burns by molten metal, red hot slags, fire, etc., while scalding with boiling or hot water leads only to superficial skin excoriations and singeing of the eyebrows and cilia. Powder explosions or lightning usually produce burns of the first and second degrees upon the skin covering the lids.

The healing of cuts, stabs, shot wounds, bites, tears or contusion of the lids is determined by the position and extent of the injury. Other determining factors depend upon involvement of the eye itself and its adnexa and the presence of infection. Accordingly, there may be either perfect healing or notches in the palpebral margin, coloboma of the lid, ptosis, ectropion, excessive lachrymation—especially when the canaliculi have been severed—symblepharon and shortening of the conjunctival sac. Erysipelas, gangrene, tetanus and rabies have also been repeatedly observed. Of general diagnostic value are emphysema of the lids, due to entrance of air from the nasal accessory sinuses in rupture of the lachrymal sac or fracture of the orbital walls, and the very gradually increasing hemorrhage into the lids in fracture of the base of the skull. The latter often causes amblyopia or even amaurosis by indirect injury

of the optic nerve. The diagnosis is established by testing the visual acuity, and by the direct and consensual pupillary reaction. Atrophy of the optic nerve will become evident after two or three weeks. Of further importance are hemorrhages into the lids accompanied by facial, conjunctival and retrobulbar hemorrhages, in consequence of stagnation of the cranial vessels caused by traumatic compression of the thorax.

Perforations of the palpebral skin by foreign bodies which have penetrated into the globe, orbit, or accessory sinus of the nose or the cranial cavity are very important. Only recently I observed a lid fistula at the inner part of the upper lid, which had been received fifteen months before while chopping wood. Several operations were required before a wood splinter, $2\frac{1}{4}$ inches long, which had made its way along the inner orbital wall, was found.

Treatment.—In uncomplicated stabs and cuts of the lids, the area is carefully cleansed with a cotton tip saturated with benzine or ether, or it is painted with tincture of iodine. The wounds are then sutured, the superficial ones by skin sutures and those passing through the entire thickness of the lid or palpebral margin by exactly appositioned skin or conjunctival sutures. The conjunctiva is sutured first. Fresh or contused wounds, or such as have been polluted by foreign bodies, are cleansed in the same way, trimmed and, if at all possible, immediately united. Older wounds of this description are best treated with a monocular or duplex bandage, saturated with a 1 per cent. solution of acetate of aluminum. The bandage should be changed daily, until the contused edges of the wound are clean. They should then be sutured. In order to encourage the rapid growth of epithelium in polluted contusions and burns of the lids, pulverized silver nitrate is recommended (Bier-Baruch).

According to Bier-Baruch's directions, powder sufficient to form a thin layer is applied over the wound. It is then slightly pressed upon it with a cotton tip. Any powder in the vicinity or on the parts where epithelium has already formed is removed by a light air spray. The bandage is changed every two, three or four days, as required. In the intervals, "in order to take full advantage of the irritation attained, in favorably progressing formation of epithelium," a simple sterile compress is applied.

Wounds located at a distance from the lid margin often heal without suturing. For palpebral hemorrhages and emphysema of the lids a moist compress is indicated, for burns and corrosions of the lids an ointment compress (byrolin, ung. hydrarg. oxydat. flav. $\frac{1}{4}$ - $\frac{1}{2}$ per cent.). Should a faulty position of the lids and the intermarginal seam, or an extensive defect of the lid substance, persist, regular massage of the scar with vaseline may effect a correction; if not, the treatment should be referred to an ophthalmologist versed in plastic operations. Such operations, however, are indicated only after complete consolidation of the scars.

For removal of traumatic ptosis, comp. p. 355.

As to the lachrymal sac, external injuries demand a bandage or suturing, according to circumstances. Fracture of the bones surrounding the sac must in most cases be established by the X-ray. As a rule, however, it is only their consequences,—occlusion of the lachrymal apparatus with polypous proliferations at the mucosa of the sac, etc.,—that call for actual treatment (comp. p. 121).

5. BURNS AND CORROSIONS OF THE CONJUNCTIVA AND CORNEA

Burns caused by molten lead, iron, boiling fat, etc., or corrosions by soluble lime compositions, solution of soda, aluminate of soda, ammonia or acids,—as, for instance, sulphuric acid,—may cause serious injury by extensive, dense scars of the lids, conjunctiva or cornea, and suppurative disintegration of the cornea with subsequent suppurative iridochorioiditis. A corrosion by pure or not highly diluted, inorganic acids, especially muriatic, sulphuric or nitric, may also cause opacity of the lens. Fehr and others observed the local effect of crude nitric acid twenty hours after corrosion: the lens of both eyes was distinctly opaque, the cornea having remained transparent.

Artificial fertilizers, especially superphosphate, contain very injurious ingredients—acetic phosphate of potash and anhydride of phosphate, which are easily soluble in the lachrymal secretion.

Rubbing the eyes with a finger to which particles of these fertilizers are adherent introduces them into the eye, causing a destruction of the cornea. They should be handled with caution.

Conjunctival catarrh due to slight ammonia corrosion soon disappears; in severe forms, a white, friable membrane will be formed and extensive traumatic surfaces, which undergo cicatrization later. There is desquamation of epithelium in a slightly injured cornea, which looks dull and often diffusely opaque.

The changes caused by burns from molten metal and corrosions by lime, etc., are similar, but much more extensive (Fig. 45).

Treatment.—In corrosions caused by ammonia, solution of soda or

FIG. 45.



Ectropion of the right upper lid of the eye after a burn.

acids, prolonged irrigation with tepid water is the first step. When ammonia penetrates into the interior of the eye, Pichler recommends rapid puncture of the anterior chamber, which is to be repeated several times. Even the most minute particles of milk of lime or mortar which have found their way into the conjunctival sac should be removed rapidly and thoroughly. The aid of a third person may be necessary and applications of cocaine ointment. The cornea, scleral conjunctiva and the everted palpebral conjunctiva must be wiped off with a firmly twisted cotton tip immersed in oil; adherent particles are carefully removed with a small spatula or spoon, care being taken not to injure the conjunctiva. The conjunctival sac is then thoroughly irrigated with cold water, particularly the region of the upper fornix, which is especially prone to harbor

lime particles. Chaloupecky thinks that after-irrigation of the conjunctiva with a concentrated sugar solution, which is a common method of removing adherent lime particles, is injurious, because of the caustic lime saccharates formed thereby. Washing with carbonic or bitter waters has been recommended, in order to transform the lime into non-corrosive carbonate of lime or gypsum. Copious quantities of oil and other fatty substances, or 1 per cent. cocaine ointment, are distributed within the eye, a protective bandage is applied, and the case immediately referred for ophthalmological treatment. Should this be impossible, atropine is instilled; later a 1 per



Symblepharon after lime burn

cent. iodoform ointment with an addition of 1 per cent. cocaine and amyloglycerine as a constituent. In my experience, this accelerates the dissolution of the scab, and is better than sublimate ointment in reducing the scars and adhesions of the fornix of the inferior conjunctiva and those between the tarsal and bulbar conjunctivæ, *i.e.*, symblepharon (Fig. 46). Applications of heat also assist in desquamating the corroded parts. The adhering parts should be repeatedly separated by drawing off the lids carefully, and moving the globe in the opposite direction.

Insoluble potassium carbonate in the tissue can probably be dissolved with ammonium tartaricum (see below).

The symblepharon operation, which in most cases has to be per-

formed eventually, may offer great difficulties. The later it is performed the better the chances of success. In lighter cases I have found it best to sever the adhesion and cover the resulting tissue gap and the neighboring conjunctiva, which has been detached from its base, by transplanting a non-pediculated skin flap, as I have described in the *Münchener med. Wochensch.*, 1897. With this method I obtained permanent success in a case which had been vainly operated upon three times. There was at first very troublesome desquamation of the epidermis of the transplanted skin, but under daily instillations of a 1 per cent. solution of soda (in equal parts of glycerine and water) this improved after a few months, and finally disappeared. At the present time, more than twenty years later, the motility of the operated eye is unimpaired.

Skin burns which are confined to the epithelium generally heal in a few days, with a bland ointment and a protective bandage, even when the entire area is involved. But deeper corrosions very often lead to cicatricial pterygium, or to necrosis of the entire cornea, with prolapse of the iris and staphyloma, or, directly or indirectly, to phthisis bulbi. This may occur in cases which at first promised well. Cyst formation in the palpebral part of the lachrymal gland (dacryops) from lime burns occurs less frequently. Cysts due to other traumas—blows, etc.—are generally located behind the bulging lid under the conjunctiva, and also on the sclera.

In the lime corrosions of the cornea, a dissolution of the resulting non-transparent opacity (potassium carbonate and aluminate) should be attempted by tepid eye baths of 10 per cent. neutral ammoniated tartar, —a mixture of ammonia chlorate 10 per cent. and tartaric acid 0.05 per cent.; this applied once or twice daily for half an hour, and the treatment continued for some time. As it is important that the solution should come in contact with the cornea, an ointment of ammoniated tartar should be applied, followed by an occluding bandage. This procedure should increase the resorbent effect.

Conjunctival shortening and adhesion between the palpebral skin and the bulbar conjunctiva and cornea, persisting after these corrosions and burns, cannot always be removed. However, satisfactory cures have been attained, even in difficult cases, by plastic operations. These measures, though, should be deferred until the cicatricial process is complete, and there is no undue irritation in the eye.

6. CUTS, STABS AND TEARS OF THE EYEBALL

The healing of cuts and stab wounds of the globe, especially those puncturing the coats of the eye, depends in great measure upon the position and nature of the wound. It is assumed that the globe has not been destroyed or much vitreous lost. Septic wounds of the cornea are

generally more benign than penetrating wounds of the sclera with exposure of the vitreous, because the latter does not tolerate protective agents well.

In perforating tears of the globe (from arrows, etc.) the wound is very often infected. But even an infection of the vitreous may be arrested, and sometimes a satisfactory cure effected, so far as vision is concerned. The patient's relatives should always be warned that sympathetic inflammation threatens the uninjured eye, unless competent aid is given at once.

One may have to content himself with an external cleansing of the wound and a temporary bandage, without touching or pressing the globe in any way.

In injuries of the globe, the iris often participates by prolapse, tears, or direct dissection. Perforating wounds often give rise at a later time to so-called serous and pearl cysts of the iris, by the scattering of corneal epithelium or epidermal cells.

Even when these injuries take a less irritating course, they may later lead to blindness and a gradual shrinking of the globe (phthisis bulbi).

Traumatic emphysema of the cornea, due to injuries by needles, scissors, etc., is characterized by a shining, punctated, more or less circumscribed opacity of considerable size in the region of the wound. It is of no importance, unless the cornea is infected.

Softening of the globe is very rare in infancy and childhood, and is characterized by a reduction of intra-ocular tension (hypotony) and shrinking of the globe. It is usually due to disease of the sympathetic nerve of the brain, and may be accompanied by lachrymation, photophobia, a feeling of pressure in the eye and violent neuralgia.

A minute corneal fistula may give rise to similar symptoms. Thus, an exploding mineral bottle caused a perforating corneal injury which, with the exception of a very fine anterior synechia, apparently healed smoothly. But the injured eye remained soft, without ciliary injection or evidence of iridocyclitis. Enucleation was considered, in order to protect the other eye, which showed distinct capillary hyperæmia of the optic disc. But a Westien-Zehender loupe revealed a minute corneal fistula of the anterior synechia. Galvanocautery promptly closed.

Dissection of the lens capsule by a stab or cut is nearly always followed by complete lenticular opacity—traumatic cataract—due to the entrance of aqueous or vitreous humor. When the wound is very small and closes rapidly, and other conditions are favorable, more or less vision may be preserved. Otherwise, and provided the injury of the capsule was aseptic, asthenopia will persist, on account of the high hypermetropia resulting from the resorption of the lens. This excludes a correction by glasses for near vision, because the retinal images of the

aphakic eye are so small as to preclude fusion with those of the uninjured eye. Often, however, a glass for distance will be tolerated. Should the other eye become blind, the damage is not great, provided the cataract is uncomplicated and has healed kindly. These patients retain over two-thirds of their working capacity.

Treatment.—If the injury to the lens is recent, the conjunctival sac should be most carefully disinfected (see above), and mydriatics, combined, if indicated, with atropine-cocaine-adrenalin, should be instilled. An ophthalmic surgeon should be consulted at the earliest possible moment, as special treatment is necessary. This is the more urgent, as in extensive injuries to the capsule the anterior chamber becomes so shallow, from rapid swelling of the lens, that secondary glaucoma may develop, demanding the immediate removal of the swollen lens. Treatment by a specialist is still more important in traumatic infection, because, if the uvea becomes involved, dense adhesions may form between the lens or its remnants and the iris and ciliary body (cataracta accreta), which very often leads to gradual blindness and shrinking of the globe.

Aside from the local remedies mentioned, constitutional treatment by inunctions of mercury ointment, together with large doses of salicylic preparations, has often yielded favorable results.

Posterior cortical cataract of traumatic origin, following a dislocation of the lens in its capsule, occurs even without a lesion of the zonula of Zinn or rupture of the capsule. It is usually caused by the action of foreign bodies upon the peripheral areas of the lens, or by contusion of the globe. In the incipient stage, the posterior layers of the lens show a stellar opacity, slightly permeable to light, whose rays extend rather uniformly in all directions from the posterior wall, leaving the periphery free. This opacity either increases slowly in intensity and extent, or it clears up again. The clearing-up process may take place after some time, with considerable improvement of visual acuity, which, previously, was noticeably reduced. In some cases the opacity practically disappears, but the chances are that the lens will become quite opaque after two or three years.

These traumatic opacities of the lens, which may also be transitory, are therefore important from a medico-legal point of view.

In the operative removal of a total cataract resulting from such injuries the surgeon must always take into consideration the possibility of a separation of the posterior capsule, and guard especially against prolapse of the iris.

7. CONTUSIONS OF THE EYE

The ocular changes resulting from contusions vary widely.

Hemorrhages, limited to the lids or the subconjunctival tissue, are soon absorbed. The separation of the endothelium of Descemet's membrane and the uniform gray, diffusely turbid swelling of the cornea oc-

contusion, caused by the pressure of the fingers if a swimming bandage was put on the eye too early or too tight. The extensive described dislocation of the ciliary process which may cause injury of the ciliary body, was observed in a case reported in p. 196, nearly always seen with a more or less intense disc-like opacity, when the opacity is extensive. Such an opacity is removed by application of heat, massage, or pressure and later massage. The opacity is still more intense when a certain degree of inflammation of the cornea, such as follows immediately after dislocation of the globe. Tears of the iris sphincter, which are not easily visible, will not disappear. Traumatic mydriasis, due either to an imperceptible discontinuity of the constrictor pupillæ or to peripheral paralysis of its nerve—iridoplegia—is often favorably influenced by repeated instillation of pilocarpine-morphine-physostigmine (2 per cent., $\frac{1}{2}$ per cent., $\frac{1}{4}$ per cent., $\bar{a}\bar{a}$). Such treatment should be carried out every evening before retiring, for a period of several weeks. In one case of distinctly demarcated detachment of the root of the iris (iridodialysis), a cure followed the use of mydriatics, such as atropine-eucaine-eupiprenin ($1\frac{1}{2}$ per cent., 2 per cent., 1 : 1000 $\bar{a}\bar{a}$), and the application of a duplex bandage to prevent pupillary reaction. Usually, however, a centripetal dislocation of the detached part of the iris will persist, with a straight-lined course of the corresponding sector of the pupillary margin.

Contusions limited to the ciliary body cannot be demonstrated with lateral illumination, nor with the ophthalmoscope. But the blood cysts in the ciliary body prove that hemorrhages in the tissue may occur. In one case the diagnosis was assured by the spontaneous bursting of the cystic wall and the consequent demonstration of numerous hæmatoidin crystals in the floor of the cyst, there being no iris at its site (traumatic coloboma of the iris). In a second traumatic case I was able to show by diaphanoscopy that the "tumor of the ciliary body" was a pseudotumor. Several experienced specialists had declared the cyst to be a melanocœroma of the ciliary body, and urgently recommended removal of the eye.

A contusion, inflicted anteriorly, sometimes causes a slight annular opacity of the anterior lens capsule, which very often requires artificial dilatation of the pupil in order to be seen (Vossius). One case was associated with traumatic papillitis. There also occurs a cataract-like opacity, originally confined to the anterior cortical layers, a tear of the capsule at the equator or posteriorly. Luxation and subluxation of the lens often cause secondary glaucoma, by stretching and tearing of the suspensory ligaments, particularly in the first weeks. If the help of an ophthalmic surgeon is not immediately availing, the severely injured eye is soothed by rest in bed, instillation of pilocarpine-morphine (2 per

cent., $\frac{1}{2}$ per cent., $\bar{a}\bar{a}$) and a bandage. This may permanently quiet the eye. But everything directly or indirectly tending to increase the intra-ocular pressure or to disturb the lens must be avoided.

Subluxation of the lens is always serious, even when the condition remains about stationary. It leads either to a refractive myopia and irregular lenticular astigmatism, both of which, as a rule, are not susceptible to correction, or there will be two indistinct retinal images, producing an effect similar to that of a pupil with congenital ectopia, part of which contains lenticular substance, while another part does not. The aphakic part of the eye—with the exception of emmetropia—is now considerably hypermetropic; the lens-containing part is myopic, due to an increased curvature of the lens, as a consequence of abnormal tension of the zonula. This gives rise to monocular diplopia. The disorder of vision is less serious in simple axial torsion, where the oblique position of the lens produces astigmatic distortion of the retinal images.

The operative removal of a lens displaced into the anterior chamber must be left to the ophthalmologist. The extraction of a freely movable, dislocated lens is a serious matter, owing to the almost unavoidable loss of vitreous. I therefore prefer to make an iridectomy in subluxation. With a well-behaved patient this is possible without untoward happenings. After the local anæsthetic has been instilled repeatedly, the iris is brought out with a small blunt hook which the operator turns backward around the pupillary margin. This little piece of iris can then be cut off without loss of vitreous. A lens dislocated into the vitreous better be left untouched. The condition is more dangerous still when, with simultaneous rupture of the sclera, the lens is displaced under the conjunctiva or drawn outside of the eye. An injury of this kind generally causes blindness, owing to the vitreous hemorrhage or retinal detachment.

Uveal irritation and inflammation often persists, especially if the scleral tear closes slowly or if the wound is infected. Such cases often require enucleation later, for the protection of the other eye, even though the initial course was apparently favorable. Simple subconjunctival ruptures may cause sympathetic irritation after many months of absolute quiescence, rendering the same prophylactic step necessary. Contusions from violent thrusts or blows may not only cause concentric tearing of the sclera about 1–2 mm. away from its margin, but also prolapse of the iris and ciliary body into the wound, followed by traumatic aniridia and softening and shrinking of the globe.

It is a fact of medico-legal interest that diffuse parenchymatous keratitis of constitutional origin, especially the typical congenital syphilitic form, may also break out after contusions or an injury by a foreign

body. The second eye becomes similarly affected after a short time, which Vossius attributes to a kind of sympathetic irritation.

It is still undecided whether an eruption of iridocyclitis, which in the final analysis is attributable to a constitutional affection, such as tuberculosis, syphilis, etc., may be caused by a blunt injury to the eye.

All ocular injuries occasioned by contusion, even though the bulbar conjunctiva is not visibly torn, demand immediate treatment by an ophthalmologist after the eye has been thoroughly and carefully irrigated with a non-irritating disinfectant like mercury oxycyanate 1.0 : 3000.0 or H_2O_2 (1 per cent.) and a temporary bandage has been applied.

(Edematous opacity of the retina (commotio retinae) occurs after contusion of the globe. It is usually located at the site of injury, or frequently on the opposite side, or it appears as a semicircular opacity around the macula. It disappears in the course of a few days, leaving no trace.

But a powerful blow may cause dropsical degeneration, intense shrinking of the chorioid and centre of the retina, and sometimes even a perforation of the macula which appears as a light red spot about the "size of a disc," surrounded by a turbid gray area. The grave consequences of such an occurrence, which may reduce the visual acuity to the recognition of fingers in close proximity, have so far baffled all attempts at amelioration.

Isolated tears of the chorioid usually occur between the macula and the transverse section of the optic disc, either concentrically or equatorially, or both, and very often occasional development of pigment, and thick scars, which cause permanent visual disturbances of marked degree when there is simultaneously a retinal tear, or a serious hemorrhage into the retina and vitreous.

First aid consists in bandage and rest in bed; further treatment belongs to the ophthalmologist. In mild cases I also advise immediate resort to mydriatics in order to ensure absolute quiescence of the retina and chorioid. This is continued until the wound has healed.

Cuts and stabs in the retina may run a more favorable course, in the absence of serious complications, such as a loss of vitreous.

A patient, sixteen years of age, suffered a nearly lineal tear of the chorioid, running through the macula lutea. Yet under this treatment central visual acuity was almost completely restored. This treatment suggested itself to me by the favorable course of the case of scleral tuberculosis mentioned on p. 228. In asthenopia and blindness due to fracture of the base of the skull, causing compression, contusion, injury or concussion of the optic nerve in the optic canal or in the posterior

part of the orbit, X-ray pictures and functional hearing tests are much more important, as in the early stage the ophthalmological findings are usually negative.

A violent contusion of the eye, which in any case demands careful treatment, may also produce retrobulbar compression of the optic nerve. In one case where there was only a slight effusion of blood into the anterior chamber, I succeeded in restoring the normal field of vision with a disappearance of the central scotoma and improvement of the impaired central vision from 6/20 to 6/8 and from Niden 4 to Niden 2 for near vision, by adopting the following course: absolute accommodative protection of the eyes, mild diaphoresis, brucin injections into the temple and fibrolysin injections into Tenon's space.

Unilateral glaucomatous atropic excavation of the optic disc, which I have observed after injury to the base of the skull with almost complete blindness of the affected eye, is probably caused by a cicatricial retraction of the orbital part of the optic nerve and sinking of the lamina cribrosa, in consequence of a contusion or tear of the optic nerve in the optic foramen. The deep marginal excavation of the pale disc, kinking of the vessels, venous stasis, pulsation of the arteries, and a peripapillary halo-like zone, correspond to the ophthalmoscopic picture of glaucoma. But hypertony, the typical glaucomatous displacement of the vessels to the nasal side of the disc, and the reduced motility of the iris which occurs with prolonged persistence of glaucoma are absent.

8. INJURIES OF THE ORBIT

Injuries of deep orbital structures are usually due to fractures of the base of the anterior cranial fossa. Subconjunctival hemorrhage of the lower fornix, extending from backwards forwards, frequently appears from one to three days later. The globe rarely protrudes. Contusion of and hemorrhage in the optic nerve, or its intervaginal space, caused by rupture through the optic canal, is a matter of serious import. It generally leaves behind unilateral and sometimes bilateral amblyopia or amaurosis, which, like the visual disturbance in direct cuts and stabs of the optic nerve, is, almost without exception, incurable. The functional test of acuity of hearing is of great value in making a differential diagnosis.

Traumatic paralyses of the ocular muscles following knocks or blows on the head, contusion of the skull, injury of the orbit by foreign bodies or muscle tears, may give rise to a variety of symptoms. These are usually caused by basal or orbital lesions, and may also be associated with injuries to other cranial nerves, the facial, optic, trigeminus, sympathetic, auditory or glossopharyngeal.

Paralysis of the ocular muscles is rarely due to an indirect trauma. Yet Partenheimer and others observed complete unilateral paralysis of

the motor oculi after a fall upon the buttocks, the head never touching the ground.

A few more details may be in order. Purely orbital fractures located further anteriorly are apt to be accompanied by motor disturbances of the eye. Total ophthalmoplegia, an effusion of blood, and associated with anæsthesia of the orbital contents, point to a lesion of the superior orbital fissure, while ptosis, paralysis of the trochlearis, and anæsthesia of the forehead (supraciliary nerve) point to fracture and dislocation of the anterior part of the upper orbital wall; paralysis of the inferior oblique muscle indicates fracture of the lower medial orbital wall.

The *prognosis* in all these types of paralysis depends, above all, upon the sequelæ of the injury. It is relatively good in an orbital lesion and in paralysis due to intracranial conditions, when not attended by concussion of the brain, fracture of the skull, injury to the nerves, or tearing of the muscles. According to G. Müller, it is noteworthy that in the course of a traumatic oculomotor paralysis the fibres supplying the vertical muscles are very often the only ones affected, or remain more paretic than the other branches. Although the prognosis of an isolated paralysis of the trochlearis is more favorable, its healing proceeds more slowly, because the gliding apparatus of the trochlea, which has been damaged by blood effusions and subsequent resorption, can resume its function but slowly.

The entrance of air after fractures of the anterior orbital segment, or after incision of the lachrymal sac or of the nasal, ethmoidal, frontal or superior maxillary sinuses, when added to disturbances of the lachrymal apparatus, produces a palpebral, orbitopalpebral, conjunctival or orbital emphysema, in which the lids and globe become doughy and often markedly protrude. The entrance of air is due to a fracture of the septum between orbit and nose, and heals in a short time without evil consequences, if bandages are applied and instructions not to blow the nose are obeyed.

The sequelæ of stabs into the orbit may be the severing or tearing of ocular muscles and their insertions, severing of the optic nerve at the apex of the orbital, with immediate, incurable, permanent blindness. The ophthalmological findings are nearly always negative at first. A progressive paling of the optic disc becomes visible only after three or four weeks. If the stab takes a cerebral direction, the optic tract is likewise endangered.

Septic stab wounds of the orbit may give rise to panophthalmitis. Injuries of the globe or orbital tissue, caused by foreign bodies, may cause *traumatic tetanus*. This often ends fatally, especially when the foreign body has not been removed upon opening the orbital abscess. As compared with grave infiltration of cellular tissue in orbital phlegmon,

the inflammatory manifestations in tetanus are relatively slight, while the motility of the eye is early and markedly restricted—the beginning of tetanus of the orbital muscles.

R. Hack describes a left-sided paralysis of all ocular muscles along with the first branch of the trigeminus, with continuous formation of ulcers in the totally insensitive cornea, which set in two and a half years after an accident. The condition was supposed to be due to fracture of a bone near the superior orbital fissure and subsequent formation of callus with lesion of the nerve ends in that locality (?).

Treatment.—Wounds of the ocular muscles should be sutured as soon as possible. Infected orbital cuts and stabs, causing palpebral swelling, chemosis of the conjunctiva, dislocation and protrusion of the globe, violent pain and fever, demand evacuation of the pus by incision, followed by gauze packing and moist compresses. Such treatment may prevent blindness and fatal meningitis, through the spreading of the orbital suppuration or inflammation to the superior orbital fissure. Shot wounds should be left undisturbed as long as possible. In all suspicious injuries of the lid, globe and orbit, it is advisable, in addition to incision and removal of foreign bodies, to inject tetanus antitoxin immediately (Merck 20 antitoxin units=4 c.c. of the quintuple tetanus antitoxin Hoechst). Narcotics should be given if the symptoms warrant.

Inasmuch as traumatic ocular paralysis often rapidly undergoes a complete cure, after having remained stationary for months, operative interference for correction of the anomalies caused by the muscular paresis should be held in reserve. This refers to children as well as adults. It is well not to operate before the lapse of nine months or a year.

Retraction of the globe: Traumatic enophthalmus is caused by a depressed fracture of one or more orbital bones and an enlargement of the orbit by the escape of orbital contents toward the deep parts, by cicatricial shrinking of the retrobulbar tissue and fixation of the globe, and possibly by tearing of the bulbar fascia and lesion of the orbital muscular bundle. This condition is irremediable. In marked hemorrhage of the retrobulbar tissue enophthalmus may precede exophthalmos. Atrophic disturbances may set in after the trauma. This interferes with the motility of the eyeball. Groenholm tried to remedy this disturbance by massage and passive movements, by means of forceps hooked to the conjunctiva. This causes the upper lid to descend, simulating a slight ptosis, which is probably caused by a traumatic sinking of the orbital opening. An improvement may sometimes be effected by suturing the superior palpebral levator in an anterior position, according to the method which I employ in congenital ptosis.

Pulsating exophthalmos, which is generally the result of a traumatic communication between the cavernous sinus and the internal carotid,

has been successfully treated by Kreeke by ligating the common and the pulsation had subsided. Otherwise ligation of the common carotid is to be considered, since repeated digital compression rarely removes the vascular communication. In one case I effected a cure by electrolytic atrophy of the ectatic orbital veins.

9. SYMPATHETIC OPHTHALMIA

The pathogenesis of the sympathetic inflammation occurring only after perforating injuries of the eye, and which is followed by an insidious inflammation, continues to be a matter of controversy. According to Mackenzie-Leber-Deutschmann, the infectious agent ascends from the infected eye in the sheaths of the optic nerve upward to the chiasma, runs around the latter and then descends as a kind of meningitis to the optic sheaths of the second eye. As a matter of fact, fever, delirium and epileptiform paroxysms have been observed in sympathetic ophthalmia, also transient bilateral impairment of hearing of considerable degree and even permanent deafness, without inflammatory changes of the external or middle ear being noticeable.

Ramsey and Sutherland found, as the first symptom of an impending sympathetic ophthalmia, enlargement of the blind spot, slight myopia and hyperæmia of the disc of the second eye. Optic neuritis, resulting in total or partial pallor of the disc, has been repeatedly demonstrated without implicating the second eye. Like Rervan, Sutherland and Wingenroth, I have observed optic neuritis, in spite of enucleation, associated with pronounced recurrent papillo-retinitis and material impairment of visual function.

L. Berlin and Römer regard sympathetic iridochorioiditis as a specific metastasis brought about by the general circulation.

According to Ziem, Motais and Gilbert, the metastasis also takes place through the venous anastomosis between the two orbits—across the bridge of the nose, through the veins of the nasal septum, the end ramifications and connections of the ethmoidal veins and the sinus circularis. The nasal cavity is likewise intimately connected with the orbit, as well as with the cranial cavity.

F. v. Arlt and H. Müller maintain that the inflammatory irritation is transmitted by the ciliary nerve. According to Schmidt-Rimpler this occurs by the action of bacteria and noxious chemical substances in the organism, which are harmless in a healthy organ, but cause a sympathetic inflammation in one damaged by irritation of the ciliary nerve. According to Bach, inflammatory symptoms in the other eye will abate, if caused by free irritating substances in the general circulation; but they will continue to develop until they have reached a degree of inflammation which cannot be checked by the enucleation of the eye.

originally affected, if the general circulation harbors bacteria that find their way into the eyes.

Elschnig thinks that spontaneous iridocyclitis of one eye may lead to sympathetic ophthalmia, and, indeed, binocular iridocyclitis generally takes a course that is distinctly unfavorable to vision.

For the clinical picture, compare pp. 231 and 232.

According to Burk, uranin reaction (compare p. 234) after an injury "furnishes certain signs as to whether sympathetic inflammation of the other eye is impending or not."

The eruption of the sympathetic inflammation does not occur until the end of the second week after the injury, and in most cases after from four to eight. After many years of latency it may be transformed into phthisis dolorosa, or a fresh trauma may be the starting point of inflammation.

Prodromal signs of an impending sympathetic ophthalmia are gradual impairment of vision, persistent irritation of the injured eye, lachrymation, photophobia and asthenopia of the second eye, especially in bright light, slight injection of the second eye and catarrhal swelling of the nasal mucosa, especially on the nasal half adjoining the injured eye.

The *prognosis* in children, and especially in debilitated, undernourished girls, is not very favorable. Provided the exciting eye has been enucleated, the sympathizing eye has the best chance of being saved if the disease has not become more than a chorioretinitis, in which numerous yellowish plaques are scattered throughout the middle and equatorial areas of the fundus. On the other hand, fibrinous plastic uveitis leads almost without exception to pupillary occlusion and seclusion, and to blindness, even though the initial stages be slight.

Treatment.—As to prophylaxis, the remarks made on p. 331 hold good, with the additional statement that injury even of a blind eye may lead to sympathetic ophthalmia. The only sure preventive is enucleation, whereas neurectomia optico-ciliaris and evisceration of the globe are unsafe. The decision as to surgical intervention is clear after the irritation of the injured and nearly or totally blind eye has lasted for more than four weeks; it is not so clear when vision still remains.

After sympathetic ophthalmia has begun, enucleation will no longer be effective. Enucleation of the injured eye can, therefore, be advised only when it is blind, and not when there is still vision. Indeed, it has occurred that the sympathizing eye was destroyed, while the function of the injured eye was maintained. In the treatment of sympathetic iridocyclitis, mercury inunctions and, above all, salicylate of soda or aspirin in large doses (up to 10 Gm. and more per day) have proved advantageous.

While Gifford and Widmark were not afraid of causing toxic symptoms with this treatment, Stock states that aspirin, even when given in conjunction with bicarbonate of potash, causes not only anorexia, but often delirium and, as a result, such intolerance for the remedy that it must be discontinued. Nenzosalin is better tolerated. It is marked by Hoffmann-Laroche in packages of twenty tablets of 0.5 Gm. each. Stock prescribed up to 20 Gm. daily, 4 Gm. to be taken every two hours, followed by a draught of milk or water.

De Ridder obtained rapid improvement with 0.6 salvarsan in a case where subconjunctival sublimate injections and mercurial inunctions had proved ineffective. During the treatment he also injected pilocarpine subcutaneously, and some time afterward subconjunctival sublimate solutions once a week.

Bernheimer arrested a grave sympathetic inflammation in a child with positive tuberculin reaction by tuberculin injections.

DISORDERS OF SPEECH AND PHONATION IN CHILDHOOD

By

DR. M. NADOLECZNY, Munich.

I. PSYCHOLOGY AND PHYSIOLOGY OF THE DEVELOPMENT OF SPEECH

AN exact knowledge of the development of speech in children is an indispensable foundation for understanding and correcting lingual disturbances. I shall follow in the present pages the explanations laid down by Meumann and the excellent monograph written by C. and W. Stern on "Kindersprache," Leipzig, 1907. Later observations by Gutzmann have also been made use of.

I. PRELIMINARY CONDITIONS FOR THE DEVELOPMENT OF SPEECH

In order to produce so complicated a performance as the human language, certain preliminary conditions must be present in the physical and psychic development of a child which will enable him to acquire the first rudiments of speech. Although the ability to hear readily suggests itself in the first place, the other organs of special sense should not be left out of account, because, in the language of Kussmaul, "hearing is not an indispensable regulator of articulation," although it doubtless plays a very important part in the development of speech.

Meumann distinguishes four kinds of development:

- (1) The acustico-optical;
- (2) The motor-kinæsthetic (development of the muscular sense);
- (3) The ideomotor (development of motor independence);
- (4) The general physico-mental.

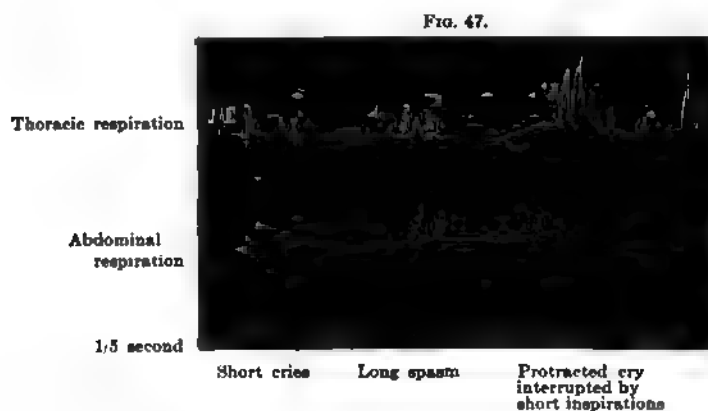
(1) **The Acustico-optical Development.**—The new-born infant is considered physiologically deaf, a statement which Kutvirt denies. Gutzmann observed an "acoustic reflex" as early as within two weeks from birth.

The fact is that an infant does not distinguish sounds before he is two months old. From about that time the unconsciously soothing effect of the mother's or nurse's voice may be observed, more still that of musical sounds to which an infant is more susceptible than to words.

At the same time the influence of visual perceptions should not be underestimated. Gutzmann thinks that the optic impulse to speak is

even more powerful at times than the acoustic. It is a certain fact that an infant observes and tries to imitate the movements of the lips as well as any other expressive movement (mimic). He will even touch the lips of those talking to him.

(2) The motor-kinæsthetic development is shown by as yet very imperfect attempts at controlling the lingual musculature and by their further development under the influence of the sense of touch, therefore under kinæsthetic control. We know little that is definite about the details of this chain of developments. The first sound is the reflex cry which illustrates the most primitive physiological process of phonation. A sound will be perceived in the widely opened air column without any articular motion, which is formed by changes in respiration and constitutes the first stage of the future lingual respiration. Careful



Infantile cry-curve. Incoördinated movements of respiration. *J*, inspiration, *E*, expiration.

observation of quiet respiration with the pneumograph shows that the thoracic respiration slightly precedes the abdominal one, and is therefore primary. Inspiration and expiration behave alike in this respect. In crying, however, the thoracic movement considerably predominates, and expiration after a long cry lasts considerably longer than inspiration: a mode of respiration which fairly corresponds to the future lingual respiration. The short cries, following the protracted one, do not show the same peculiarity, but the short cry-curve has already a greater resemblance to the speech-curve of respiration. The uniform course of the protracted cry-curve is disturbed by the fact that the musculature of the entire organism assists in the cry (Fig. 47). The expiration is carried out "with all fours," as Niemeyer has it, first with incoördinate movements, which in the third year are replaced by well coördinated movements, when talking. The timbre of the voice, when crying, has the predominating crowing sound of the vowel "a" at first; at about the fifth week this changes to the expression of pleasure or displeasure,

according to the infant's wants, so that the mother may, for instance, recognize from the tone-tint and its intensity whether he feels hunger, pain, etc. In this way the expression imparted by the cry is full of information.

(3) It is far more difficult to understand the **idiomatic development** of how unconscious sounds change into conscious. It may be assumed that the positive result of the expressive movements as a reaction to endogenic or exogenic irritations is associated with the latter. In this way, a previously simple motor discharge would gradually change into a conscious utterance of sounds.

(4) **The general physico-psychic development**, which is also a preliminary condition, cannot be analyzed in detail in the first year of life. Normal progress during that period requires the development of concentration sufficient to observe the surroundings and expressive motions. This is also required for the infant to observe and control at a later period his own utterances. He must also be able to retain to some extent impressions he has received by the organs of special sense (eye, ear, touch). The congenital impulse to imitation should be no less lacking than its first rudiments. This may be designated as the child's psyche. It is just this internal factor which will later form one of the strongest impulses in learning to speak.

These preliminary conditions are by no means preconceived and invariable, but will perfect themselves more or less in the course of general lingual development.

2. PRELIMINARY STAGES OF LINGUAL DEVELOPMENT

The actual lingual development is preceded by preliminary stages which, according to Kussmaul, may be classed as three, but which cannot be rigorously separated as to time of appearance.

(1) The preliminary stage of spontaneous lallation appears as early as the fourth to seventh week in some infants. This stage of development has been characterized by Taine as follows: "The child acquires the raw material of speech." He plays with his speaking apparatus as he does with his arms and legs. This lallation must be regarded as a pure product of an impulse to talk, as a simple motor discharge. Wundt rightly assumes a heredito-physiological disposition for this phenomenon. The function of the sensory sphere, however, is not entirely excluded, because the infant experiences tactile and acoustic sensations and thus learns in time to control his own sounds with those two senses, accentuating his acoustic perceptions on the very ground of his own sounds. Thus, in the course of this period, there will develop the senso-motor association of spoken sounds and perceived sounds. "The child imitates himself" (Stern).

As early as the fifth, but usually between the seventh and eighth weeks, the infant commences to express his emotions by smiling, exultation and lallation; various and sometimes complicated sounds of satisfaction escape him, especially after a meal; the first consonantal elements of the language are brought into connection with vowels, which are no longer heard as crowing explosives as in crying, but sound lower and softer owing to the slower approach of the lips. Gutzmann distinguishes the soft "pleasure sound" which he first observed in the tenth week, from the harsh "sound of displeasure," pointing out that adults, too, employ both ways of expressing their sentiments (Fig. 51).

The first, or primitive, sounds are produced by narrowing the air column between the lips, between the tip of the tongue and the alveolar margin, between the back of the tongue and the palate, and between the base of the tongue and the posterior faucial wall. The sound current is here dulled and fuses into a friction sound, until at last there is an explosive. There is no difference in principle between vowel and consonant, even in the stage of development. There are at first cooing sounds, called vomitive sounds by Kussmaul, which disappear later in the German language (although they exist in other civilized languages); then follow labial and lingual sounds, also clicking sounds which in civilized languages are but the rudimentary expressions of regret, astonishment, etc. These clicking sounds are not difficult to produce. They occur by simply opening an occlusion of the articulation tube during inspiration, while the expression of explosive sounds requires actual muscular effort. These sounds (clucks) are recognized in the regular Hottentot language and therefore indicate an inferior degree of culture.

In the second preliminary stage of lingual development, at the age of eight or nine months, the infant first commences to imitate sounds, then words, heard from others. It is not certain how far this is done with conscious intelligence. However this may be, we are not justified in distinguishing between lingual imitation with and without conscious intelligence, as marked by special periods. The auto-imitation in lallation, a form of spontaneous talking, precedes the imitation of strange words and sounds. If sounds are uttered to the infant which are taken from his own baby vocabulary, he will be able to imitate them relatively early, say at the age of two and a half months, although with difficulty. Although the sounds may be perfectly identical with those he has formed himself, yet the timbre of the adult voice differs considerably from the infantile. Besides, in lallation the infant follows his own inclination, while in imitating the sound heard from others he will have to accommodate himself to a strange perception. On the other hand, there is a congenital impulse to imitation which is by no means limited to lingual sounds. Gestures, grimaces, unarticulated noises, songs, and timbre

of the adult voice are imitated with evident delight. In this way, infants gradually arrive at the stage of echolalia (see p. 447). The intentional repetition of sounds which are not included in the "baby-talk" still causes great difficulty, and the attempt is often a partial or total failure. This may explain why attempts at teaching sounds often meet with great resistance. For this reason a period of physiological "acoustic dumbness" may be interpolated in the development of speech, during which, however, the infant continues to absorb the language unawares. The development of lingual understanding progresses far more rapidly than the ability to talk, so that the number of understood sound-complexes soon far exceeds that of self-uttered sounds.

Before proceeding to examine the development of lingual understanding, the physiology of sound, as it exists in this period of imitation, may be briefly described.

The difficulties of intentional imitation of sounds do not lie so much in the necessary power, but in the skill required for coördination. Here is the principle of the least physical exertion coming into its own. The least difficulty is offered by the vowels, such as AH, O, AY, while EE and OO require a little energy. This difficulty may be assumed to correspond to that of narrowing the air column. With consonants, and consonants connected with vowels, the situation changes. Here, numerous and variable movements coöperate with each other; in changing from M to AH, for instance, the velum palati has to be raised. For this reason, M as a humming sound appears earlier than in connection with a vowel. Again, baba, which does not involve any motion of the velum palati, is repeated earlier than mama. Friction sounds require the retention of the position of articulation for a certain time and are therefore more difficult than explosives. On the other hand, simple vibrating sounds, at least those of the lips (labial R), are not difficult to form, because the latter, following the law of gravitation, are passively moved by the air. The conditions for narrowing and widening the oral fissure are simpler at the lips than in the second or third place of articulation, where the tongue is compelled to perform complicated movements in order to utter an explosive sound. Thus, it is not surprising that the complicated hissing sounds (S, SH, Z, X) are developed at a later stage, because the place of articulation, required for their production, has yet to be formed, quite aside from the fact that the teeth have to coöperate therein. Indeed, the process of dentition should not be overlooked in the physiology of the development of speech. The L-sounds, too, are formed later and are produced by children with the tip of the tongue behind the dental process of the inferior maxilla, similar to R, while adults, in pronouncing L, raise the tongue to the alveolar process of the upper maxilla. This may probably account for the substitution of

L for R, and *vice versa*, in the baby language, as well as in the language of uncivilized races, in the Sanskrit, etc. The pronunciation of the K-sounds (K, G, NG) is not only aggravated by the complicated movements of the tongue, but also by the undeveloped muscular sense at the fundus of the oral cavity, thus explaining their relatively late enunciation. Parallel instances are again found in the language of semi- and uncivilized races. The possibilities of imitation are further aggravated by the fact that the movements required for the production of these sounds cannot be seen by the infant.

This will give an idea of the difficulties of imitating various sounds.

It is clear that these phonetic difficulties considerably increase with a complicated sequence of sounds. Their repetition, therefore, cannot be expected in this period, which is confined to the simple alternation of consonant-vowel-consonant, with a preference for the repetition of like syllables.

As was mentioned before, the period of the third preliminary stage, that of understanding words, coincides with the second stage. Based upon the logical reasoning of adults, a similar function was formerly attributed to the infantile mind, of which it is unquestionably incapable during the first years (for instance, for the formation of general conceptions, etc.). Preyer has done away with these speculations, while Meumann has shown the way for understanding this part of psychic development. We must imagine the first dawning of understanding speech to be primitive in the highest degree, as "associations of the most rudimentary kind," ranging between the timbre of the mother's voice and certain soothing affects (satisfying the craving for food). When an infant, upon hearing the name of things or persons,—that is, an acoustic impression,—responds by a movement of the head or eyes toward the object named, this would be rightly considered as an understanding of the spoken word.

This stage is illustrated in the literature by the classic example of Lindner. Lindner's son, when twenty weeks old, was repeatedly taken to a clock, the syllables "tick-tack" being simultaneously pronounced. After a few days the infant looked toward the clock whenever the syllables "tick-tack" were pronounced. Meumann attributes this to a sound-association, by which an infant's attention is directed to a known noise, which is followed by a movement in that direction. He correctly adds that space orientation always appears early.

As a second example may be mentioned an observation of Sigismund, because it was intended to use the same for the demonstration of a hypothesis in regard to the early development of general conceptions. When one year old, the child was shown a stuffed woodcock, the word "bird" being pronounced at the same time; he then looked at an owl

standing in a different corner, thus showing that he had an abstract conception of a bird and recognized the characteristic signs of these animals, etc., a performance which considerably older children are unable of bringing about. Meumann attributes this performance likewise to a simple association between a known object-picture (owl) and the searching look for the same, caused by the positive, though not perceived, similarity with the other bird, with which the enunciation of the word "bird" had nothing whatever to do. It was simply a recognition of a known object and the association with the movement of the eyes, perhaps even a primitive distinction for which, however, there is no proof.

It will thus be seen that these examples are attributable to a simple acustico-optico-motor association and to a certain understanding of the gestures. In assuming interpretations of this kind, it will always be difficult to avoid errors.

The same holds good for another infantile action, in which the roots of lingual understanding are to be looked for. This is based on the gestures employed while playing or training, and which an infant acquires when about one year old. Gestures made on hearing phrases like "shake hands," "how tall is the little one?" "make please," are well known. They imply requests and are accompanied by expressions of pleasure on the part of the adult and by a smile or some sound indicating pleasure on the part of the infant. The same words and gestures are constantly repeated, the infant's hands are led so as to incite him to imitation amounting to passive movements. This kind of training, which is facilitated by the impulse to imitation and which at first has only a mechanical effect, is, as Meumann has shown, of great importance for the development of lingual understanding. With its aid, the infant enters into the habits of adults, he learns to distinguish the timbre of different voices, expressiveness, information and demonstration, partly by the modulation of the voice and partly by observing the accompanying gestures. At a later period, however, he will recognize the significance of a sign or designation by the mere gesture.

Since the desire of possessing and receiving predominates in an infant, he will absorb impressions of this kind with great facility. His emotional and volitional lingual understanding is therefore less keen than the associative-intellectual. This applies not only to the understanding, but also to the first stage of actual talking, in which the expressions of will and wishes predominate, as will be shown presently.

The causal problem of infant language, as follows: Does an infant learn to talk by imitation, or is it a congenital trait?

It can, therefore, be summarized, of himself; is he simply within owing to a derivation of the

preliminary stages, as described above, will show that C. and W. Stern are right in saying that speech develops by the convergence of external and internal factors. The doctrine of independent speaking will furnish numerous proofs for this assumption.

3. THE FIRST INDEPENDENT TALKING

The infant has run through the preliminary stages explained above: lallation, senseless imitation and primitive understanding. The latter phase must still be associated with a motor utterance of his own in order to render "communicative" talking possible. This progressive phase usually sets in with infants of the educated classes after they have attained an age of about one year and nine months, no precise observations being available as yet for children of the uneducated. The further progress is just as little uniform as in the preliminary stages: periods of progress alternating with periods of apparent arrest during which, however, the understanding of speech may rapidly develop, while primitive sounds will be entirely forgotten. The same undulations which dominate the psychic development, and almost all phases of development, are also present in the development of speech.

According to the Sterns, children require the assistance of intonation and gestures in order to arrive at the "threshold of understanding" for a few words, while the great majority of words are still unintelligible at that period; other words are reached at the "threshold of speaking," but not that of understanding. The word "threshold" is intended to mean "any kind of irritation which just commences to exert a psychic function." Words used for intelligent speaking must have crossed both thresholds, which, however, does not mean that they must have reached the full meaning of adult language. On the contrary, they are still very far from it. Newly acquired sound-complexes may be identical in their outward appearance with those used by adults, but they are at first used by the infant to express his wishes, desires and sentiments.

The Sterns go fully into the details of the conditions which govern the selection of these words and are the basis of future progress. The psychological conditions, for instance, include the child's interest, which is more attracted by movements and actions, or by infantile requirements, than by non-moving objects. Physiological conditions are centred in phonetics, in which the facility of forming sounds plays a part. Again, the surroundings and the manner of talking to an infant contribute to the selection of words.

In this way, the "one-word sentence" originates, consisting of a single word, supported by intonation or gesture, which according to grammar, but not according to sense, is an interjection or a substantive. The period of the one-word sentence lasts for about six months.

The fact that the same word is used for widely related or partly similar things led to the idea that the infant formed "preliminary conceptions" (Romanes), or "rudimentary conceptions" (Ament), or "conceptions of great generalization" (Preyer).

1. Meumann, instead of starting from our logical language, which is totally foreign to the infantile intellect, raised the question: "What does a child mean with his first words?" He showed that an infant does not form any conceptions at all, but gives expression to his wishes and desires, or does not mention an entire object, but merely some part or phase of it that happens to strike him. (Emotional-volitional stage of wish-words of Meumann.)

An example, cited by E. Schulte, may illustrate this. An infant understands by "cap" all kinds of hats, covers, etc., and, by uttering that word, expresses a wish to have that object. Meumann does not look upon the word "cap" as an expression of generalization, but as a wish-word, uttered for the purpose of playing with that particular object, or putting it on his head, which, however, has nothing to do with the conception of the word hat.

The first lallation is probably an expression of joy at the ability of phonation caused by noticing some object or act, similarly to our spontaneous lallation as an emotional accompaniment.

All the first infantile words evidently bear the character of wishes, just as at a later stage the first questions express a desire. The numerous first words in "baby-talk" are considered by Meumann to be sound-imitations of strongly felt impressions and for this reason these exclamations as well as the words formed at a later period have the grammatical character of verbs and interjections.

Accepting Meumann's interpretation, regarding the first word as an expression of will and sentiments, it will not be found difficult to understand why a baby exclaims "hot" on being brought into contact with water that is either too hot or too cold. He does not express any particular perception, but merely an unpleasant sensation, or a protest.

The intellectual understanding of the language does not commence until the child begins to refer to special things or acts in his vicinity and the character of sensation is replaced by the designation of that object or act.

The Sterns assume that the one-word sentences are from the first not entirely free from a fragment of expressive character. Idelberger thinks that words at the beginning of their intellectual use are uttered on one occasion as the expression of sentiments and wishes and on another occasion as a designation. Meumann thinks that designation results from confining a wish-word to definite single objects. There is a great probability for the correctness of Sterns' assumption that certain known

symbols are reproduced on observing a similar object. This would be combined with sensation on the basis of an association of ideas. According to the direction in which the infant's interest lies, many wish words would apply to eating and drinking, while many affective words would refer to situations, coming and going.

2. It may also be assumed that the first words of designation are purely associative, and for this reason Meumann calls the second stage of lingual development associative-reproductive. The difficulty of interpreting the first words of designation and their use, has led to the hypothesis of a logical formation of conceptions, which Meumann declares to be wrong. He explains his position as follows:

A concrete perception excites the infant's attention. This perception is associated with a word, and that word is reproduced by the frequent repetition of the same perception. If the infant has perceived two phases of the same as yet unknown object, both phases will be associated with the same word. An infant does not understand the function of a word as a symbol with a certain meaning. The mechanical reproduction of his word takes place on recognizing a concrete perception. The psychic part of the baby's performance is therefore a slight one.

Romanes's well-known example is used by Meumann to explain his associative-reproductive word formation: An infant has seen a duck in the water and exclaims "quack" when seeing any other bird, insect, water, or a coin after he had observed an eagle on a coin (Preyer).

"Whatever strikes the attention on the enunciation of a word is associated with that word and embodied in the vocabulary"; hence the same word for bird and coin, after the presence of an eagle has struck his attention, which, of course, is an entirely illogical formation of word-values. The word "quack," however, has been associated with the phase of perceiving a "duck in the water," with anything that has wings or flies, and with water. This phase of perception reproduces the word which is used for insects or liquids, since the infant has not yet assimilated their inherent properties. He therefore does not abstract a meaning, nor does he form general conceptions. Similar concrete perceptions reproduce the same impression, and the similarity of apparently heterogeneous things is so much greater, the slighter the accuracy of the perception and the more phases strike the attention, which latter, of course, is yet slight. The other unknown phases are overlooked. He knows and denominates only what he knows. This explains the changeable significance of words, of which the Sterns say in similar words that the points perceived become carriers of the symbol, which is not always the same, the associate component likewise participating. It should be kept in view, however, that the symbols as such are not abstracted, but merely form the perception as a whole. That what strikes the

attention most becomes the fixation point for the designation, the more so as a baby's sphere of knowledge is incomplete. The importance given to a certain phase of a perception and the recognition of the same phase in other subjects often simulate accuracy of observation.

3. A greatly superior lingual performance sets in with the period of one-word sentences if the child uses his word intelligently, showing that he is reaching the stage of logical lingual intelligence. In order to recognize the transition from the primitive words of the first stage to intelligent expression, to recognize the penetration into the function of language not merely as an expression and communication, but also as a means of designation, it is necessary to recapitulate the genesis of symbols. The factors in natural and conventional formation of symbols have in part already been mentioned. It is now necessary to consider them in their entity. Starting from the psychological, fundamental fact that internal comprehensions are converted into movements, the Sterns derive symbols in the first place from natural gestures and sounds. They divide the first sounds of lallation into two classes: the homogeneous or onomatopoeic, as for instance bow-wow or tic-tac, and the heterogeneous, such as *mammie* and *daddie*, in which a direct internal relation to the object is lacking. At a later stage these words become conscious property and are used for communication and then for designation.

On the other hand, sounds assume conventional symbols under the important influence of imitation which entirely dominates the further development of speech in the first three years.

This imitation, which at first may be involuntary, is soon controlled by the will. The imitated sound is senseless in its origin and may even remain so for some time, according to individuality. It is then reproduced in echolalia, until the imitated sound-complex becomes understood and is used as a symbol.

A peculiar, but by no means rare, occurrence is observed in the "mediate form" of imitation. A word heard by an infant at some time, suddenly reappears on a similar occasion without having been purposely uttered, and the infant will recognize the word from the situation by association and reproduce it, although before he could not be made to do so by immediate request.

In how far the imitation is phonetically correct depends upon various factors, such as hearing, intelligence, remembering, the lingual motor function and the phonetic difficulty of the word. The intended meaning of those symbols, which at first differ from their conventional signification, renders a logical understanding difficult for adults and leads to misapprehensions, while it is easier for us to understand the meaning of the expressions of will and desire from intonation and gesture.

But since the definite meaning of our words must again and again influence the infant's speech, he will in time be compelled to adapt his conception of the words to that of our own. This coercion of adaptation is aided by his daily wants and the failure of his efforts in trying to have his wishes gratified by wrong expressions. In this way he is led to select his words properly. Meumann looks upon this stage as the initiation into intelligent speech, the progress of which is greatly influenced by accuracy of perception, acuity of attention, and memory.

He further points out that, as a matter of course, the number of acquired associations divide themselves into groups of associated part manifestations, within the sphere of which the conception regularly connected with a word takes superior rank, without the child being aware of its intrinsic logic.

In this way definite expressions will be formed as early as about the fifteenth month—at first for those concrete objects with which the infant is most concerned. This is designated by C. and W. Stern as the "stage of lingual substance." The meaning of these words has, therefore, been acquired from the immediate surroundings. Articles of nutrition, toys and living objects, such as come within the sphere of his desires and interest, as well as his own requirements, such as eating and drinking, are those which he first learns to designate. The comprehension of words is still influenced at a later period by individuality. At that time, objects are elucidated as "individual conceptions" (Stern), in the formation of which personal inclination, *i.e.* the animus, participates, as it does in all psychic processes of the child.

The intellectual enunciation of a word is not complete in the stage of one-word sentences. The conceptional and lingual progress continues, so that full sentences may perhaps not be uttered before school age.

Words in the logical sense, not in that of one-word sentences, will of course mature until a later time, since no child acquires speech on grammatical principles. Considering the truth of this fact, it may now be opportune to follow the further development of words.

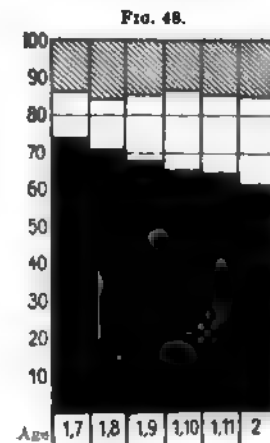
The logical process of abstraction does not commence until about the fourth year of life. At first the fact is recognized that many objects have a number of points in common and are therefore grouped together from that point of view, although not as yet under a general conception. At this stage of "plural conception" (Stern), the child, for instance, knows quite a definite tree, whether in nature or in picture books; to this is added the knowledge of another tree, etc.; but if there is a clump of trees, he will not put them under one group. It is not a far step to arrive from this plural conception to general conceptions. Thus, toward the end of the fourth year abstractions will be understood: a performance which a former psychological period has attributed to the second year.

The further development of thought depends upon the development of speech. "The formation of general conceptions and the establishment of self-consciousness, as against the former passive existence, are only possible in man within and with the language" (Sterns). Certain laws for intellectual development have been laid down by the Sterns for the acquirement of the various categories of speech. In the "stage of lingual substance" none but objective words are acquired; in the "stage of action" active processes are perceived; in the last or "relative stage" the prominent points of external things are abstracted." (Fig. 48). These authors also demonstrate this sequence for other performances of psychic development.

The external development of words is also dependent upon a number of factors, both endogenous and exogenous. When a child has become conscious of the symbolic meaning of a word, he



Quantitative increase of vocabulary of Deville's daughter from one year seven months to the end of the second year



Distribution of word categories during the development of speech of Deville's daughter in per cent. Black, substantives, white verbs, hatched: other words except interjections

will naturally look for new words, for the names of various objects, thus increasing his vocabulary considerably, especially at the end of the second year.

The classification of existing words which has been compiled by numerous authors will illustrate the rate (Fig. 49) and logical form of the development of speech. The statistics of words used (Gale) exemplify the sphere of juvenile interests, the chronological examination of various words (Ament, Idenberger) illustrates the gradual understanding of their significance. All these statistics distinctly show that the number of acquired words increases in periods, thus again showing irregular progress.

As in the formation of symbols, the lingual character of the words is changed. According to Meumann, a short space of pure echolalia seems to be still present in the imitative period. At first, words of onomatopoeic character predominate and are parts of the objects. Later, however, imitations of adult language prevail, including the conventional symbols, but, owing to a certain imperfection of infantile ability, the imitations are insufficiently developed and are almost unrecognizable as new formations. But it has

been shown by Preyer, later by Wundt, and in a particularly interesting case by Stumpf, that these words are only seemingly new formed. It has already been mentioned how stubbornly young children are apt to retain the use of their own words, offering a certain resistance to all attempts at correction. How far this tendency may go is well illustrated by Stumpf's careful observations on his own son, who obstinately persisted in his own terminology of mutilations up to his fourth year. Similar cases were also observed by Hale, Moore and Taine. One afternoon Stumpf's son suddenly discarded his way of talking, which was only intelligible to the initiated, and used conventional language, which he had evidently well assimilated in the meantime. Stumpf explains the origin of this mutilated language by the (acoustic) crude material of expression used by ourselves. The impulse and pleasure of the child to make himself understood, supported by the surroundings, led to the further development of speech, which does not strictly partake of the character of new formations. At a later age, however, these new formations do occur. The motive for doing so is attributed by Stumpf to "certain analogies of perception and relations which are shared by the impressions of various senses owing to a certain similarity or other occasional circumstances." "The origin of the expressions themselves, however, is mostly shrouded in mystery owing to the individual nature of the analogies."

The imitations sometimes furnish but mere fixation points for spontaneous development and treatment of the language under the compulsion and need of expression. This, according to Stern, partly takes place by association. Such words are, therefore, but analogies and only rarely etiologically permissible; they imitate the phonetic expression for the designation of known objects, as, for instance, "potgun" instead of "shotgun."

Faultless repetition of words, however, is not always easy, even where progress is not purposely arrested by wilful mutilation. There are not only sensory and motor difficulties, but also mistakes owing to inaccurate memory. Faulty perception prevents correct reproduction in spite of an undoubted endeavor to acquire it, leading to mistakes of enunciation or physiological stammering (see p. 396). Not even in adults does the perception of words take place sound by sound, but is determined by the predominating component parts. The principal cause is no doubt a disproportion between the desire to speak owing to insufficient motor ability and exercise. There may be improper model enunciation, as for instance where there is an only child; or insufficient care, as in uneducated and low classes, which will retard the acquirement of speech. On the other hand, exaggerated demands during this period may work great mischief.

Phonetic changes are characteristic for this period. The principle

of selecting a physiologically slighter exertion, which has already been referred to, may also be a factor in the case, as for instance the substitution of EE for AY, S for SH, L for D or R, and dentals for gutturals. The predilection for duplicating syllables may be explained in the same way or by ataxia, but according to Meumann indistinct hearing also comes in for a share of the blame. Part of the phonetic changes is attributed by the same author to a central adaptation to the preceding innervation and to a certain tendency of persisting in the motor function which has already been initiated, while at the same time the necessary attention is still lacking.

These mutilations may be divided into the following classes:

Omission of letters, as, for instance, feat for feast. Strictly, however, these are not omissions, nothing being eliminated that was present, the sounds not pronounced simply not having existed. Similar to omissions are phonetic changes, such as tub for tough.

Metathesis is also a favorite mutilation, such as tantern for lantern, Bezzelin for Zeppelin.

Contaminations are less frequent, such as jacking for jacket. Assimilation is worthy of special notice. This, according to Stern, may be of a general nature, if all sounds resemble one that was preconceived. More interesting is assimilation which results from anticipation and consists in changing a preceding sound in the sense of the following one. This is metaleptic or regressive assimilation, as in tlantern for lantern, nannon for cannon. In children, however, this occurs less often than proleptic or progressive assimilation, in which persistence in innervation is the deciding factor, while anticipation is absent, as, for instance, penpolder instead of penholder. These examples are taken from six-year-old children whose lingual development had been somewhat arrested.

The grammatical part of word development corresponds to the three stages of the Sterns that were mentioned above. Pure substantives, at first without inflexions, are learned in the stage of lingual substance, verbs in the stage of action soon play a considerable part, and then come the adverbs, adjectives, etc., in the stage of relation. Inflexion is acquired in the third year and simultaneously in all categories.

4. FORMATION OF SENTENCES

When discussing the development of words, it was already pointed out that the "first words" have the character of sentences. The same refers to lingual comprehension, because a child understands but few words of a coherent sentence—the principal ones, from which the rest of the sentence is combined with the important help of gesture and intonation. The grammatic development of infant's talk has been first examined in detail by the Sterns and Gheorgov. According to these

authors, sentences containing two or more words are formed before the middle of the second year.

Sentences of two words consist of subject and object, verb and object, verb and adverb, subject and verb. For instance, "papa birdie" would mean "papa is bringing a bird"; "Lulu there" for "Lulu is there." In sentences of more than two words there is no grammatical arrangement, one word simply following the other.

As early as toward the end of the second year compound sentences are formed, first again for the purpose of expressing wants or sentiments. It is a characteristic peculiarity that the thing that is not wanted is placed first: "chair no no lap," would mean: "I do not want to sit on the chair, but on your lap," thus forming an antithesis.

Indication of wants is followed at the end of the second year by statements or questions, but their expression is still in the form of wishes and are therefore questions only in name. The statements refer to events which have excited the child's interest.

At the end of the third year, the first secondary sentences are formed, but without any connecting particle: "cut paper funny," would mean: "the paper I have cut looks funny." The relation between the various parts of a sentence develops next. As these questions at first always bear the character of a wish, those inquiring about a name or a desired object are formed earlier (in the third year); the same refers to questions to which an affirmative answer is desired, but a negative one apprehended. These are still wish-questions.

It is not before the end of the fourth year that the celebrated why-questions appear upon the scene, but still under the mask of wishes, such as: "Why may I not," etc.? These are followed by attempts at finding a logical coherence.

Finally, time-questions are embodied in the sentence.

The sequence of the words in a sentence is again regulated by imitation and individuality. The word it is intended to emphasize is often placed first, the psychological subject preceding in thought the psychological object. At a later time the child adapts himself to our own form of language according to individual conditions.

As the development of sentences progresses, new words will be formed after the manner mentioned at the end of the preceding chapter. Gale's statistics show that verbs are used much more frequently than substantives, although the latter considerably preponderate in the first year. The Sterns have explained how the infantile language gradually assumes the grammatical form of the language. They found that, so far as verbs are concerned, the infinitive and imperative are acquired first; later, in the beginning of the third year, the past and the future tense. Adjectives expressing taste and temperature precede those ex-

pressing abstract ideas. Here again antithetic words, like light and dark, have seemingly the same value, as they are merely intended to express a sensation, and not its quality. Differentiation in colors is first expressed in the third or fourth year. Counting commences at the age of two in a primitive way by collecting a number of similar objects in the sense of plural perceptions. Mentioning a figure, usually 2, then simply has the meaning of several. The correct use of figures, as applied to definite, concrete objects, is acquired much later, often not before school time.

The negation "no" frequently precedes the affirmative "yes" and is often used for it. The first "no" is then intended as a general dislike of everything, which is then followed by "yes." The personal pronoun "I" is used later by some children than by their brothers and sisters. Instead of "I" they prefer using their own first name, and this is always the case with the first born. The language of a six-year-old child is at best only phonetically developed, hardly ever in a grammatical sense. Further progress beyond that stage belongs to the realm of the school.

5. DIFFERENCES IN DEVELOPMENT

Language does not develop alike in all children. The beginning of independent infantile talk may be delayed until after the second year. Progress, too, is quite irregular. It may require longer or shorter periods, according to the extent of the vocabulary of different children.

The Sterns attribute this variation to internal and external reasons. The latter include the social stratum to which the new-born child belongs. The manner in which parents, etc., care for the child in the pre-talking period is of importance, and his toys and pictures participate in first impressions. The influence of older brothers and sisters is often decisive in the manner and speed of acquiring the language. The Sterns relate a peculiar case, how twins ardently assisted each other in the development of speech at the end of the second year, and the dominating influence was exercised by the otherwise inferior infant. The only child of an educated couple usually enters school with a greater vocabulary and a better mode of expression than others, which, however, does not necessarily mean greater intelligence. The influence of the dialect spoken by various nurse girls is well known. The same authors mention the further fact that children rapidly acquire a foreign tongue at the relatively late age of four years, if they happen to be taken to a foreign country, for the reason that the word-sounds of their own language had not yet been firmly established and associated with logical ideas.

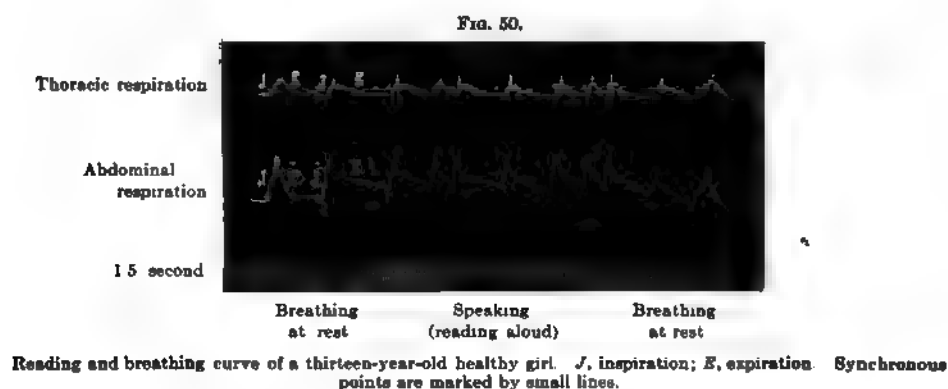
The internal conditions are usually summarized as the individuality of the infant, but, aside from isolated cases, exact knowledge on this subject is still wanting. However, the fact is established that **girls**

learn talking more rapidly than boys, possibly because they undergo more rapidly psychic and physical development and have greater adaptable and imitative faculties. Boys are duller and more obstinate (Preyer) and incline to a pronounced individuality, which is noticeable at an early time. They attempt to express comparatively much by small lingual efforts. Probably it was not an accident that it was a boy who formed the subject of Stumpf's communication in regard to the peculiarities of handling the language.

Thus, there is always an effort at spontaneous or inherited impulses and faculties which are partly peculiar to family influence and partly to the individual, to intentional or unintentional external influences, or, in the language of the Sterns, "they are the result of a constantly active convergence."

6. PHYSIOLOGY OF THE COMPLETE LANGUAGE

The above explanations have furnished a foundation for understanding disturbances of speech, and there is still to discuss the physiological part of the fully developed juvenile language. Assuming that the difficulties of articulation have been overcome and examining the



various physiological factors that enter into lingual performances, the following points will present themselves. Respiration, which does not conform to definite rules in the first years of life, adapts itself to enunciation. Since a comparatively large quantity of air must be slowly, but uninterruptedly, distributed at expiration over a large number of words or sentences, more air is required during speaking than when silent. Respiration must therefore be deeper and there should be no impediment in the air passages, so that the air current may pass through the open mouth, without constricting the tube of articulation. Thus, there is a distinct difference between the uniform, automatic nose-breathing when silent, consuming but little air, and the irregular, deepened mouth-breathing with an increased consumption of air. The thoracic curve is no longer synchronous with the abdominal curve:

while the latter is already sinking, the former is still ascending. Thoracic breathing, therefore, predominates when speaking (see Figs. 50 and 54). The vocal cords, while breathing slowly, are moderately apart, leaving a triangular space (the vocal glottis) between them, but, while speaking, respiration causes the glottis to open to its maximum width through rotation of the arytenoid cartilages. The gap between the two vocal cords thereby assumes a quinquangular shape, and the cords themselves disappear entirely, or nearly so, underneath the vocal bands.

As to the voice itself, it has already been mentioned that there are various ways for the voice to set in. The voice should always set in, and it generally does, with a soft and low sound (Fig. 51). The hard sound (Fig. 52) is reserved for the expression of sentiment, notably of dislike (see p. 360). Whispering causes the vocal cords to approach

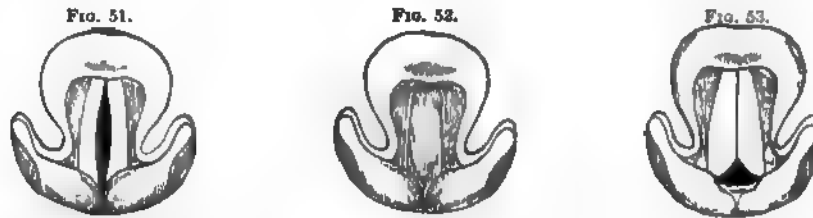


FIG. 51.—Diagram of a glottis at the onset of a soft sound, the infantile form being considered.
 FIG. 52.—Diagram of a glottis at the onset of a hard sound, before protrusion of the vocal cords.
 FIG. 53.—Diagram of a glottis, while whispering.

each other, leaving a triangular space between them, the respiratory glottis, through which the air escapes partly, while speaking. This causes the low friction sound which always accompanies whispering (Fig. 53). For the normal formation of the various sounds, see pp. 361 and 397.

This exhausts the physiological signs of speech, except modulation. This depends upon various accents. The musical accent imparts to the voice the characteristic psychic expression by modulation of the sounds between syllables, words and sentences. It differs according to age and sex. The dynamic accent effects quite independently of modulation a more or less pronounced distinctness by the force of the muscular exertion of articulation. A third factor is the temporal accent, which influences the musical accent by the duration of the sound and determines to a certain extent the rhythm of the voice.

II. LINGUAL ARRESTS OF DEVELOPMENT

BEARING in mind the importance of the preliminary conditions of lingual development, it is intelligible that their impairment or absence must result in a relative or total arrest of development. The best known factor in this respect is the influence of deafness. However, blindness, disturbances of the motor-kinæsthetic, general physical and psychic development, act as factors of arrest in the above sense, not in the psychiatric sense.

1. PERIPHERALLY IMPRESSIVE ARRESTS (TOTAL OR PARTIAL DEAFNESS, BLINDNESS)

Total or partial deafness with hearing remnants, which exists from birth, interferes with lingual development. Should it occur before the seventh year, the time a child has acquired full mastery of speech, he will become deaf and dumb. By careful instruction the faculty of speech can be retained, when deafness sets in between the fourth and seventh years. As a rule, speech will then persist after the seventh year, too. The highest limit at which deafness may occur is, according to Kussmaul, the age of puberty.

An important requirement for retaining the faculty of speech is a knowledge and practice of writing. But it may be that there is a reminiscence of latent sounds which may suddenly reappear after many years of instruction. The power of recollecting certain word categories corresponds, according to Stern, to the sequence of their acquirement.

In congenitally deaf infants there is total absence of the acoustic influence of the voice and of all other sounds. These children will continuously remain on the stage of spontaneous lallation. They will be unable to perceive their own voice and will not rejoice at it or its frequent repetition. Thus, deaf-mutes need not entirely lead a soundless life, but their sounds are phonetic discharges which have no lingual character. In very rare cases they will be able to imitate papa, mamma, bow-bow, but they have no real understanding of them. However, assistance which in normal children aids in the acquirement of speech, such as demonstrative gestures, mimic, our involuntary movements and their own tactile sense, also imparts to a deaf child a high degree of understanding utterances and sounds, so that they are often considered to be of sound hearing.

The original method of carrying on conversation with deaf-mutes is by gestures.

In sentences expressed by gestures, the emphasized and more easily intelligible word is placed first, as is the case in normal infantile talk

(see p. 373). There is also the antithetic form in which the negative part of the sentence usually precedes. Indeed, there are a great number of analogies between the language of deaf-mutes and infants. Thus, Sully cites the following sentence expressed by gestures: "Teacher, I fight, cheat, quarrel; no—I love, honor, yes," which would mean: "I have to love and honor my teacher." The gesture language is practically confined to concrete conceptions, utterances of activity and demonstration. It therefore satisfies the requirements of the stages of substance and action of baby language, but not those of higher lingual performances.

At the present time, deaf-mutes are instructed in the phonetic language, but unfortunately at a comparatively advanced age. They are instructed as in a foreign language, and not in play, as is the case in normal infants. This explains why they will seldom succeed in mastering it like a normal person, and they much prefer expressing themselves by gestures outside the school and in later life. H. Stern ardently advocates a certain preliminary education of deaf-mutes before school age (voice culture) and progressive lingual education.

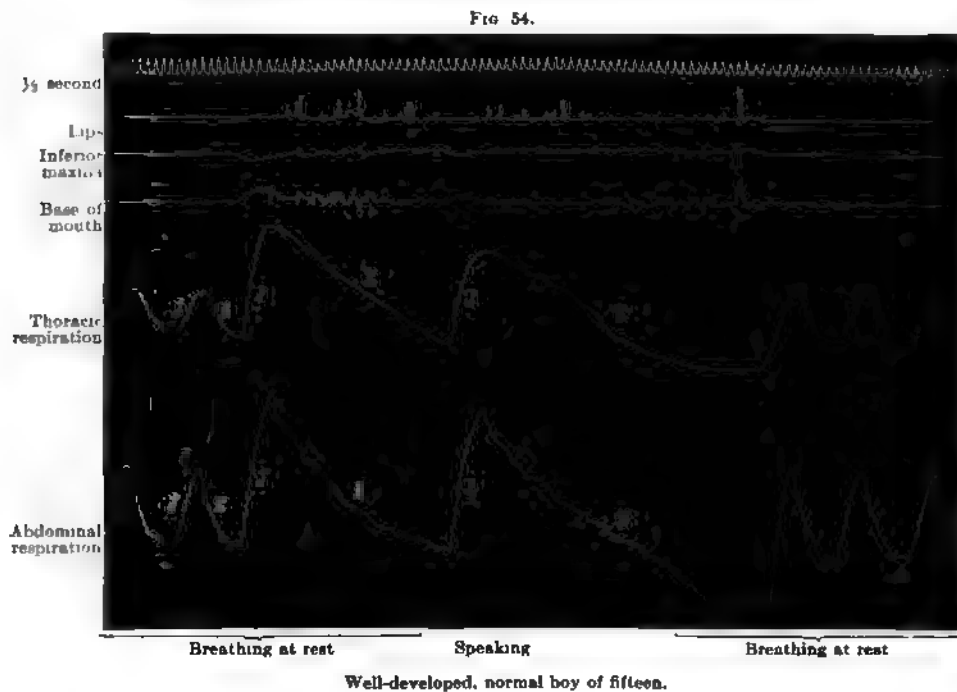
Acquired sound-language rarely attains to cultured and pure articulation, unless there are hearing remnants to mediate a certain acoustic control. There are quite a number of disturbances.

Respiration in congenitally deaf infants is not correctly coördinated with the movements of articulation, the latter being interrupted too often owing to the waste of air. Nor has the normal anachronism between the thoracic and abdominal respiration appeared as yet. If the condition escapes the attention of the ordinary observer, two other lingual defects will become so much more pronounced, which consist in defects of articulation and of lingual accents. So far as the former are concerned, they are synonymous with ordinary stammering, which will be discussed later. The voice is rough and uncontrolled by the ear, and defective pronunciation of certain sounds, notably of S, the formation of which is often aggravated by anomalies of maxillary articulation (see p. 400). Accentuation, however, suffers in every direction. Absence of acoustic control is responsible for the unpleasant modulation of the vowels and for the loss in musical accent throughout the language. Since the difficulties of articulation require a certain effort to be overcome, the dynamic accent of the voice is changed to such an extent that everybody is struck with its excessive acuity. This is always observed at an examination of the movements of articulation by abnormal contraction of the laryngeal and oral musculature. This laborious method of articulation further leads to abnormality of the temporal accent, the time of transition from consonant to vowel especially being retarded. Thus, the absence of proper coördination of lingual movements in the

characteristic deaf and dumb language is caused by insufficiency of articulation and of the various accents of speech. (Compare the two curves in Figs. 54 and 55.)

The various lingual functions of those who have acquired deafness before having fully mastered the language, including writing, will approach the normal more completely in proportion to the efforts made to retain the language.

The conditions of speech are somewhat different in impaired hearing. Here again a distinction should be made according to whether the affliction has been acquired in the first few years, or later. Its influence



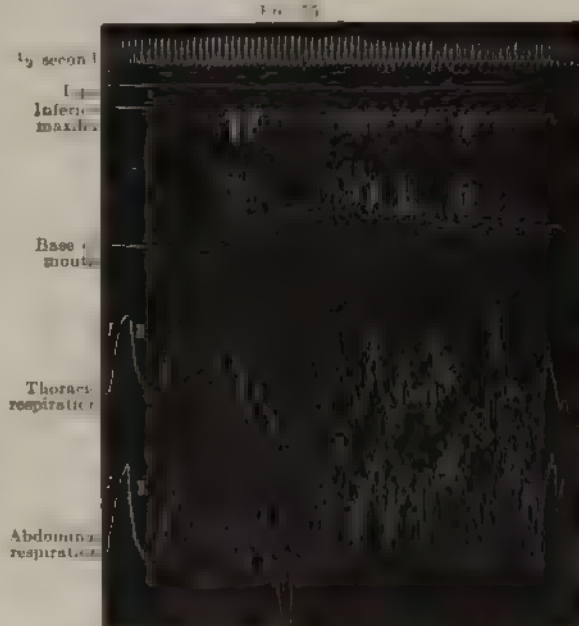
on each child is further determined by the degree and quality of the hearing disturbances as well as by the stationary or progressive nature of the affection.

It is impossible, therefore, to lay down any normal conditions for the language of hard-hearing individuals, especially as the individual ability to understand and control the position and movements of the organs of speech differs. The lingual disturbances of those with acquired impaired hearing or of acquired deafness, after having learned to speak, are therefore further dependent upon the degree of attention they have bestowed upon their organs of special sense. It has not yet been sought whether the various types of perception behave differently, but such will probably be the case.

1. In children whose hearing is impaired from early life, perception and production of lingual sounds are already injured in the preliminary stages of lingual development. It should not be forgotten that their acoustic world is not only smaller than ours, but also qualitatively different. Even assuming that all the sounds of the scale were equally reduced in regard to perception—which, however, is never the case—the sounds perceived must be of inferior quality, because all sounds do not strike the ear with equal force. Weak sounds would therefore not be perceived at all. This would not only refer to the sounds of the scale, but also to overtones as their parts. Consequently, a child with impaired hearing will not be able to control his own first sounds as completely as a normal one, nor understand as much of a conversation.

Neuert has shown that partly deaf children can still hear vowels well, but that consonants are better perceived by the eye, and that in hearing the form of articulation is perceived, but that the places of articulation are mistaken. Thus, T and K are interchanged, while in reading the places of articulation are correctly perceived, while medium and delicate distinctions as between P and B are not.

The accentuations of speech, particularly of music, are imperfectly perceived, and the same refers to the sounds in nature. In this way the acoustic attention fails to be roused and acoustic ideas are more or less absent. There is lack of impulse to psychic life and lack of material for speech. The development of the child is therefore neglected, his need for communication remains on a low level, with the consequence of a dull and uncommunicative disposition. His language is bound up with concrete ideas, because lingual progress, thinking in the language, is impossible. This means a long delay, first, in reaching the stage of one-word sentences, then in the stage of lingual substance, associated with mistakes in the syntactic structure of the language. It



Boy of thirteen, facial scars from scar fever, by bilateral ear spaces channelled out, bilateral paralysis of the facial nerve and consequent paralysis of the lips.

amounts to a disturbance called akataphasia, to which further reference will be made presently.

Children with impaired hearing are sometimes erroneously regarded as weak-minded, although it is a fact that many weak-minded children are partly deaf. Indeed, in recent times the mistake has often been made of regarding weak-minded children as of normal intelligence, after their impaired faculty of hearing had been discovered. The differential diagnosis is not always easy, since acoustic inattention of weak-minded children may simulate dysacusis.

The unskilful lingual movements of hard-hearing children impedes phonation. Difficult sounds which require rapid and complicated movements will therefore be repeated wrongly, if at all, especially if they cannot be easily read off the face owing to their complexity. Thus, the guttural sounds are sometimes absent and the S-sounds are faultily formed. In regard to S, particularly, Bloch assumes that, owing to defects in the upper sound-limit, this very high sound cannot be perceived with the ear. In short, children whose hearing is impaired since early life are usually stammerers. There is no modulation in their language, which resembles deaf and dumb language less than that of individuals who have acquired partial deafness at a later period.

2. After the language has been learned and the articulation perfected, after the words have become symbols and the language the means of thoughtful intercourse, loss of hearing still involves considerable drawbacks. The hearing ability may still suffice for that part of the language which has been assimilated, but not for learning new words. Amplification and improvement of the internal language are limited, the perceptive life is impoverished, and the acoustic control of the voice itself becomes unreliable.

According to the kind and manner of disturbance, the monotony of the voice in one hard of hearing is more or less prominent. There is no change from high to low, no difference between hard and soft tones, or it is exaggerated. The dynamic and musical accents of the language are changed. A weak voice seems to occur much more frequently than an excessively loud one; faulty sounds, or sounds too highly pitched (*i.e.* above the sounds a-d), are less frequent. According to Gutzmann, friction sounds often take the place of explosive sounds, and, owing to indistinctness of all movements of articulation, the occlusion of the velum palati may fail to act with the result of pronounced snuffing.

The treatment of children that are partly or totally deaf is of the greatest importance, the object being to use the voice as a means of promoting education and culture, and not to maintain pure phonation. So far as a cure or improvement of dysacusis is concerned, I refer to Alexander, Vol. VI.

There is no doubt that great injury is done to those partially deaf by instructing them in the same class with those totally deaf. Children with normal intelligence and a good ear for vowels, or those who become deaf after the fifth year, should be placed in institutes or schools for those partially deaf.

Special classes for the latter have been established in Berlin at the instigation of A. Hartmann. At the Institute for the Deafmute in Munich, special hearing classes have been established at the instigation of Bezold. The only private school in Germany (internes) is under the direction of K. Brauckmann, in Jena.

While the hearing organ, aside from the other organs of special sense, is trained for lingual instruction, reading off the face should also be taught at an early time, unless children have already acquired this practice of themselves. In this department of instruction, those totally deaf acquire greater ability than those who can still use good hearing remnants. The latter, however, believe that their hearing improves at the same time, since they can understand more by reading from the face.

Investigations of Neuert and Kroiss have indeed shown that three-fourths of all hearing defects are improved by using both ear and eye. The visual type of perception no doubt gains more in that way than the acoustic. Considering that those capable of reading off the face have already a knowledge of the language, they should be principally taught to read word pictures and sentences, in order to exercise their ability of combination. According to my experience, children have great aptitude in this respect.

The lingual defects of those partially and totally deaf can be removed. Rules for exercising the feeling for strength and height of sounds, by palpation on other children and by the use of the ear, have been laid down by Gutzmann. He avails himself of a duplex ear trumpet, which enables the patient to control his own voice and compare it with that of the instructor. In exercising articulation, not only the ear and the tactile sense are brought into action, but also the face, by having the exercises take place before a mirror.

Comprehensive investigations into the progress of the deaf are not yet as numerous as those into the lingual development of normal children, as described in Chapter I. Of children congenitally blind, however, we know still less in this respect. But as their eyesight is wanting, it may be assumed that they learn talking later than others.

Goldammer states emphatically that "whoever is born blind, will acquire the language later and more laboriously."

On the other hand, our knowledge of lingual development is far more complete with the deaf and blind. Ever since Helen Keller has written her "History of my Life," great attention has been paid to indi-

viduals enjoying only three senses. It was again W. Stern who compared their lingual development with that of normal children, showing that the principal difficulty consisted only in the length required, but not in the quality, although totally different senses and expressive movements (tactile sense and finger movements) were used, which have nothing whatever to do with the lingual centre, neither in regard to perception nor to reproduction.

Congenital deaf-blindness is very rare, 8 cases among 116 having been collected by Arnould. In a large number of cases, however, the condition arose before language was entirely acquired. The latter category, therefore, is almost on the same level as those congenitally deaf and blind. Bruehl found 7 totally deaf and blind among 16 cases in Nowawes, 6 cases of deafness with hearing remnants and blindness with visual remnants, 3 totally deaf with visual remnants and 1 totally blind with hearing remnants. Etiologically, one-half of the cases are due to hereditary syphilis and one-fourth to meningitis. Girls furnish 69 per cent. of the deaf and blind. Bruehl emphasizes the possibility of saving one-third by timely diagnosis and early specific therapy (syphilitic nurslings). The best known cases of deaf-blindness are those of Laura Bridgeman, who acquired the affection in her third year; Marie and Marthe Heurtin, who were born deaf and blind; Helen Keller, who lost both senses when 19 months old, and Hertha Schulz in her fourth year.

The onset of deaf-blindness first causes complete disorientation, which may also occur periodically. Such children play at night and sleep in the day. At a later period they become accustomed to a certain amount of regularity under the influence of their surroundings.

The loss of hearing does not always seem to be recognized by these patients. Hertha Schulz, for instance, remarked: "At a time when you were all able to speak." Later, a primitive gesture language sets in, in the place of which all deaf-blind children who have had the advantage of competent education are now taught the finger alphabet.

By this method, the language is acquired at a relatively late age; Laura Bridgeman and Helen Keller, for instance, at 8, Hertha Schulz at 11. For advanced education, gesture language, finger alphabet and instruction in articulation are employed, each method supporting the other.

In regard to Helen Keller, we are indebted to Miss Sullivan for further details. By using the finger alphabet, the language was acquired playingly, as under normal conditions the sound-language, and the vocabulary increased rapidly. W. Stern has shown that an intelligent deaf-blind like Helen Keller made as much progress in one month as a normal child in 6 months. Her speech development passed during that time through all the stages, including those of substance and action on an associative-reproductive basis under the impulse of desires. In the

second month a stage was reached which normal children acquire in the second half of the second year, viz., the intelligent use of the language with conscious knowledge of its symbols. At that time the first questions were asked. In the same month the first small sentences were formed with the characteristic peculiarities of the first baby sentences: parataxia, the emphasized word at the beginning and negation following after a negative word. In the third month she had reached the stage of relation and modifying words, being a higher stage of logical expression than a normal child approaches at the end of the second year. At that time reading instruction commenced. In the fourth month she was able to count up to 30 and commenced writing (squares and braille point). In the fifth month the inflexions and past tense were mastered, which a normal child would not acquire before the end of the second year. The second question-stage, including the why-questions, occurred in the second half year of her speech development, while in normal life these intellectual and no longer volitional questions are not reached before the third or fourth year. Sound-language was not acquired until the age of 10, as a "new means of communication," as Stern put it, she having otherwise completely mastered the language as a mental equipment. She does not appear to have used single sounds for purposes of designation.

Laura Bridgmann used 60 sounds mostly to designate persons, although she never acquired the sound-language. A wonderful development and degree of education, such as Helen Keller achieved, thanks to the genial gifts of her teacher and her own natural talents, probably a pronounced motor type of perception, has never been reached by any other deaf-blind child.

Hertha Schulz also acquired the sound-language, which, in Riemann's opinion even, was the carrier of her thoughts, because the deaf-blind are more inclined to think in the word-language than the deaf-mute, provided deafness has not set in at too early an age. It is a remarkable fact that she first acquired words, it having been found impossible at first to teach her single sounds. These words may be considered to range in equal importance with one-word sentences. Riemann had also to refrain from teaching her complete sentences, and substituted instead paratactic sentences of several words, as used in infantile language. They were of the pedocentric type. Psychic expressions were taught by



gestures and at first only physically understood, as for instance "loving" or "being affectionate." The sounds of the phonetic language had to be developed as in the deaf-mute. They were palpated from the mouth, combined with the signs of the hand-alphabet and then pronounced by another child and so passed on. The lingual development was aided by latent reminiscences of eyes and ears from the time preceding the affliction. Thus she remembered the color of certain objects, after she had learned their designation in her 16th year, also the music of a military band. Helen Keller even has hearing-dreams. The tactile sensations of these deaf-blinds are so delicate that a wrong rhythm will disturb them, and in the language they are associated with a number of expressions for hearing sensations, the use of which astonishes a lay person. The transplantation of expression from the extinguished sensory spheres into the language, however, should not cause astonishment as a purely lingual function. Helen Keller has described in her publication "My World" how rich the world of the deaf-blind is by the use of their delicate senses for tactile perceptions, odor and taste. The active use of the tactile sense does not differ very much from that of blind persons, but it is exceedingly sensitive for changing successive impressions, as in the use of the finger alphabet.

One of the questions to be decided is as to the kind of language the deaf-blind should be taught first and the manner of teaching it. Stern adduces good reasons for the finger alphabet, which should be acquired playfully by conversations, like the acquisition of the normal language. Riemann thinks all methods should be tried in order to ascertain the inclination of the child. He recommends a systematic procedure in the technique of instruction, especially in cases where an instructor's entire life cannot be devoted to the child, and lessons have to be given periodically. "The subjects taught and their methodical sequence should be so arranged as to mutually support and promote each other." In the instruction of Hertha Schulz he has instinctively availed himself of infantile language in part.

Instruction should be imparted under the direction of an experienced specialist for the education of the deaf-mute and the blind. This is best accomplished in an institute in which, according to Bruehl, all such blind should be placed who are too hard of hearing to follow the instructions given to the blind, and too weak-minded to follow that given to the deaf-mute. Thanks to Riemann's efforts, such an institute has been established in Nowawes, near Berlin, and affiliated with the Oberlin House. It is called the German Home for the Deaf-blind (Deutsches Taub-盲indenheim).

A last group of children whose lingual development has been arrested could be formed by neglected children whose parents think they

have done enough with providing for food and clothes, and neglect lingual incitation.

Gutzmann cites examples for this class from history, the Sterns refer to Kaspar Hauser, Wohlfahrt describes the rapid development of speech in a girl who had been entirely neglected up to $2\frac{1}{2}$ years of age, until she came under proper care. This child was entirely mute but for the one word "key," and then learned in 80 days as much as other children in a year. The Sterns assume in this case a stagnation of lingual impulses and extensive preparatory sensory work. This view is supported by the fact that in her fifth year dialect expressions appeared which she had heard in her earliest infancy. I have observed similar results in children whose lingual development had been arrested by dysacusis or motor insufficiency after the cause had been removed. Teachers in the lower grades of the public schools are well aware of the fact that "backward" children, after having entered school, show unsuspected lingual development under the influence of teachers and school-mates.

2. CENTRAL ARREST OF LINGUAL DEVELOPMENT

Central arrests of lingual development are rarely referable to sensory causes (sensory aphasia). Defects in psychic preliminary conditions (see p. 360), as for instance disinclination to talk and absence of impulse to imitation, prevent or delay the development of speech (auditory dumbness, voluntary whispering). Defects of attention lead to spluttering (paraphrasia præceps), while defective memory chiefly retards speech, aside from impairing the intellect (akataphasia). Arrest of motor development (stammering) is particularly frequent.

LINGUAL DEAFNESS (SENSORY APHASIA)

Central disturbance of speech seems to occur exceptionally in mentally normal children as a hereditary taint. Schwundt and Wagner have reported the case of two speech-deaf sisters who were thought to be deaf and dumb, but had a comparatively good sense of hearing. They repeated short sentences in a mechanical fashion without any understanding and without carrying out instructions contained therein. But when they had an opportunity of reading the sentences off at the same time, they understood them like any other deaf-mute. A boy with approximately normal hearing was able to attend a public school with some degree of success. Gutzmann states that these arrests of development of the acoustic centre of speech are exceedingly rare. His two cases had been pronounced totally deaf by ear specialists. It is important for purposes of diagnosis to notice that these children react to slight noises, although, when spoken to, do not show any understanding by gestures. They are unable to pick out an object on verbal request,

but when making, for instance, the gesture of cutting they will pick up a pair of scissors. Liebmann has described a case of psychic deafness which, however, belongs to the domain of weak-mindedness.

AUDITORY DUMBNESS (AUDIMUTITAS)

Dumbness of hearing children (*alalia idiopathica*) (Coën) is arrest of lingual development of otherwise normal or nearly normal children between the ages of 3 and 10 years, very seldom of older ones. Previous to the third year retarded development of speech is assumed, usually as a sequel to physical weakness and retarded general development. According to Schlesinger, a delay of this kind may be expected, if the cranial circumference exceeds that of the chest in children of 18 months or more. This affection has an outward resemblance to motor aphasia, without, however, being analogous to it. It is found much more frequently in boys than in girls. They understand what they are told, but do not care to repeat, converse by gestures or lingual rudiments, or, if they have commenced to talk, suddenly cease.

Causes and Origin.—According to Coën, chronic alcoholism of the parents is the prime causative factor. Gutzmann found in 37 per cent. of the cases defective lingual impulse of the parents, especially the father, which was in many cases combined with other disorders of speech. It is uncertain as to how far consanguineous marriages are responsible, and the same refers to traumas. Such as occurred at birth (about 10 per cent. of his cases), as well as other serious cranial trauma, are the most suggestive, but they suggest with the same degree of probability genuine motor aphasia. Gutzmann assumes that difficulties of articulation, caused by considerable hyperplasia of the faucial tonsils and palatal clefts, suppress the desire to speak. However this may be, children with adenoids who are very hard of hearing do not belong to this category, their hearing difficulty being sufficiently explained by the condition. According to Gutzmann, the origin of the condition may be found in arrest of the desire to talk and subsequent re-awakening of the impulse to imitation. Impulse to every kind of movement is absent in these children. Want of physical skill, and oftentimes enuresis are present as signs of pathological traits. On the other hand, it is a well-known fact that in the course of lingual development with good lingual understanding speech is present, although defective. Knowledge of the language is not always technically sufficient to meet the requirements for a considerable time, nor is the vocabulary extensive enough. Expressions of dislike may easily occur owing to these drawbacks, especially as the infantile mind is predisposed to giving way to displeasure. Shyness resulting therefrom has an inhibitive effect upon the desire to talk. This is aggravated at a later period by conscious linguaphobia, even

though the initial stages of speech had already been developed. Liebmann also considers defective attention and memory in various sensory regions responsible. As a matter of fact, disturbance of concentration, owing to unconscious internal and external deflection, is common. Based upon the doctrine of sensory types, Liebmann assumes that even the predominating sense is insufficiently developed from the above causes, while the others need hardly be considered.

Accordingly, he differentiates four forms of auditory dumbness. The most frequent is the motor form with good lingual understanding and the slightest degree of imitation. There is a mixed form with partial lingual understanding (see p. 364) and slight spontaneous utterances. These two forms belong to the group he discusses. His third form is a transition to stammering, the nature of which probably depends upon arrest of motor development and was discussed on p. 396. The sensory form (psychic deafness) is very rare. (Cases of Gutzmann and others.)

The *diagnosis* of auditory dumbness presupposes a thoroughly or approximately normal physical and psychic condition without any disorders of the organs of speech or symptoms of any organic nervous or cerebral diseases, sufficient hearing ability to understand speech, and lingual intelligence corresponding to age. The diagnosis is easy, therefore, in normal, older deaf children, who are able to follow verbal directions without reading off the face.

The *differential diagnosis* is rendered difficult by the fact that the attention of these children is easily deflected. In order to exclude deaf-mutism or high degrees of dysacusis, careful and frequent observation is often necessary, while otological hearing tests may lead to errors, as for instance by erroneous statements of parents in regard to really deaf-mute children. According to Froeschels, the tickle reflex from the auditory meatus is absent in the deaf. Sensory aphasia may be differentiated by the demonstration of lingual understanding, which, however, may sometimes be very difficult. Differentiation from light degrees of weak-mindedness is only possible at times after continued observation and examination of the function of the various senses, for the reason that perfectly normal intelligence does not often occur in the deaf-mute, the less so as absence of speech impairs the psychic function.

Examination.—Aside from physical defects, it is necessary to find out whether the child reacts to auditory irritations, recognizes noises, such as rattling with a bunch of keys, and whether he understands words and sentences. The first-named test is decided by requesting the child to point out certain objects or pictures, and the other test by verbal requests unaccompanied by gestures or looks, such as "get me that book," care being taken that the child will not be able to read off the face. The tests of other psychic functions are described on p. 443.

The following case is an example: A seven-year-old boy (Fig. 57), a twelfth child, had never been ill, according to the anamnesis. Dentition took place at the age of nine months, he learned to walk at thirteen, and the first lingual sounds were uttered at two years. Unskilful, slow movements, slight susceptibility to pain, and alternating strabismus are present. He can understand whispering at a distance of about twenty-seven feet. Spontaneous utterances occur by gestures only. He can repeat single sounds slowly, with the exception of "f," but stammers in syllables and even monosyllabic words. His attention is easily distracted, but has a good sense of orientation. Pointing to wrong objects

FIG 57



Boy with auditory dumbness. (There is a scar from a burn on the arm.)

or pictures, even though similar, will not disconcert him (shaking his head). He learns a number of words in six weeks; then, upon request, designates certain objects in drawling, monotonous tones in d-sharp and slight articulation (accents!). Tends to elisions. Rarely speaks without external stimulus. The monotony gives way to an accent of joy when imitating animal voices. There is no mimic when speaking, except an occasional closure of the left eye as an accompanying movement. Words of more than two syllables and a few sentences are learned with great difficulty. Adenotomy two years ago effected no improvement.

Course.—It may be assumed that the dumbness in this class of children will gradually be superseded by a more or less distinct

manner of speaking after an initial stage of considerable stammering, which may also change to stuttering; in any case, the affection will always cause psychic and mental impairment.

The *prognosis* is good in normal children. It is less favorable when there are defects of attention, also with defective hearing in regard to time, until independent talking will be established by appropriate treatment. This generally takes three months, but in complicated cases the degree of the other disorders has to be considered.

Treatment commences between 4 and 5 years of age and is intended to arouse a desire for repeating words. This is not done by compulsion,

but by inciting interest, telling short stories, imitating animal voices, or having games with mark-words. Books to be recommended are the following:

Any particular inclinations and desires ought to be considered. There should also be light gymnastics, demonstrations and reading. Gutzmann attaches great importance to sound pictures being accompanied by eye pictures. The first lessons are given by means of small cards containing one letter each. Simultaneously instructions are given in articulation (see p. 398), with the object of training the motor-kinæsthetic centre, developing and practising sounds, as with the deaf and dumb. Instruction in speaking should pay due regard to the phase of normal development of speech, not fatigue the child and therefore not last for more than half an hour. These children have no business in institutes for the deaf and dumb.

There is no use in performing adenotomy unless there is a demonstrable disturbance of the faucial tonsils, as the former alone is rarely sufficient. Nor is there any use in incising the frenulum. Invigorating diet and the removal of obstipation, which is of frequent occurrence, are important measures.

Prevention is not always possible. Children's musical instruments arouse interest for sounds and imitation. Exaggerated demands upon the lingual ability are to be avoided, lest children lose confidence in themselves. No attempt should be made at overcoming physiological stammering by compulsion. Forced repetition, likewise, can only do injury, incitement to speak while playing being sufficient.

VOLUNTARY WHISPERING

Causes and Origin.—Partial arrest of lingual development is called by Gutzmann voluntary whispering. It is mentioned at this juncture because, like auditory dumbness, it is attributed to disinclination to speak and is only in outward form a disorder of the voice. It is but seldom described, but occurs oftener than supposed. Gutzmann was able in some cases to trace it to early stuttering. The children observed that they were able to talk without spasms while whispering and therefore acquired the habit, one case retaining it until adult age. Accidental discovery led to a disorder of speech, which in time may cause atrophy of the vocal cords. The affection also occurs in general arrest of physical and psychic development. Here is an instance: A nine-year-old girl, physically and mentally backward, a second twin, of good family, who was not lacking in care and education, learned to walk at the age of two, and to talk very slowly at eighteen months. She is still in the second grade. The mother is psychopathic and the father is a stutterer. A sister of twelve speaks indistinctly. There is slight stammering, "sh" cannot

be pronounced and there is occasional slight snuffing. Superior maxilla prognathous. Speaks only in a whisper, but rapidly acquires better articulation. The prognosis is good and the treatment simple. Phonation takes place with the assistance of the vibratory sensation of the glottis in distinct vowel positions of the mouth, assisted possibly by the electric current (pulsating, direct current, Flatau's glottis electrodes).

SPLUTTERING (PARAPHRASIA PRÆCEPS)

Paraphrasia præceps, or tumultus sermonis, is a hasty manner of talking, in which sounds, syllables and words are swallowed, dissimulated and mutilated, a kind of pararthria (paralalia), caused by distortion of the temporal accent of speech. It occurs in both boys and girls up to the twelfth or fourteenth year, and sometimes persists to adult age.

Causes and Origin.—Spluttering may occur on single occasions in the course of lingual development, and in that case cannot be regarded as pathologic, but will be so if it becomes a habit. Defects of the peripheral organs of speech can hardly be etiologically considered, while retarded acquisition of speech and stuttering may be factors. It is due to a disproportion between a desire to talk and motor skill. It is lingual ataxia due to inattention ("a defect in the ideogenic centre," Gutzmann).

Liebmann differentiates between defects of motor attention and acoustic attention and correspondingly two different forms of paraphrasia præceps. In the one form the voice, chasing after the flight of thoughts, is not controlled by sufficient motor attention; in the other, acoustic inattention is responsible for atactic language which is unskilled in expressiveness and defective alike in diction, syntax, and grammar.

The symptomatology, therefore, is the following: Inexact formation of the various sounds is not dependent upon a definite kind of sounds as in stammering, but any one sound may be pronounced correctly at one time, and mutilated at another. All the vocal and syllabic changes which have been described on p. 373 may occur, with a predilection for elision and metathesis, also for duplications. Sometimes entire words are omitted, and as a sentence assumes a complicated form the speaker's mind becomes perplexed, he will offend against the grammatical and syntactical construction, and repeat words or exchange them for others. In the presence of strangers he will usually speak better, because he will take care of himself. For this reason, his answers to questions are usually in better form than narrations (as for instance in an anamnesis), and short sentences will be better than long ones. Repeating test sentences, unless too long, is easier than spontaneous speaking, due to the form being

in which to clothe the thoughts. Again, patients suffering for inattention fare better than those who cannot acoustically repeat. The latter are often unable to repeat the simplest sentences. Reading is likewise more or less disturbed, according to the nature of the words.

Reciting usually bears the same characteristics, as was first mentioned by Liebmann.

Liebmann found the language to be more nearly correct in reciting prayers, but in ordinary children's prayers I have observed mistakes of the worst sort. I once heard a six-year-old girl say the Lord's Prayer, but at such a terrific speed and so full of distortions that not a word could be made out.

The psychic behavior of these children immediately shows their nervousness and hasty disposition. They often can not be understood owing to the indistinctness of their utterances; they are remonstrated with and scoffed at, all of which will conduce to the exacerbation of their shortcomings.

The *diagnosis* usually offers no difficulties. In the differential diagnosis stuttering is to be considered in the first place. Pronounced atactic stuttering is certainly a similar manner of speech, but is always accompanied by spastic manifestations including the regions of the respiratory and articulating musculatures. In the presence of strangers, or when speaking slowly, stuttering or stammering will not usually quite disappear and often be associated with spluttering; stammering, however, always repeats itself with certain sounds, which is not the case in spluttering, aside from the absence of other symptoms. It always sets in at an early age.

In its further course the disorder may easily lead to moroseness, shyness and not infrequently to stuttering.

The *prognosis* is dependent upon the degree of disturbed attention, energy and intellect. Generally speaking, it is good and a cure may be expected in from four to eight weeks. It is only in very severe cases that a much longer time is required, the length of which will depend upon the individual's repeating ability.

Treatment.—The primary object of the treatment is to procure physiologically correct phonation which includes slow enunciation, if necessary before a mirror, aside from the removal of stammering. The first exercises consist in repetition of words and small sentences, followed by reading aloud first in a monotonous tone, if otherwise impossible, and then with the musical accent. In patients who exhibit an exceptional degree of acoustic inattention, Liebmann has the question repeated before it is answered. The next exercise consists in repeating the contents of fables or reading matter from books used in the lowest grade.

asked by the little patients offer the best opportunity for defects, and they should therefore be patiently replied to.

AKATAPHASIA AND AGRAMMATISM

Akataphasia (Steinthal) is a syntactic disturbance or "inability to express the flight of thoughts."

Causes and Origin.—The causes of being unable to construct sentences are various. As was pointed out on p. 372, the sentences that are formed may undergo periodical arrests and remain behind the average progress of a particular age. A frequent causative factor is weak-mindedness in its various degrees. Even normal or nearly normal children, who may be gifted with powerful powers of observation, may be subjected to such a period. It is mostly observed in children who at first were hearing-impaired stammerers, and had consequently to suffer in linguistic development. It also occurs in paraphrasia. Liebmann always found a marked attention of one or more sensory regions in conjunction with a considerable want of motor skill. Walking is also acquired with difficulty. As far as adults are concerned, Heilbronner refers the disorder to motor disturbances, having demonstrated the interference in the sensory sphere.

Liebmann claims (for children) an additional absence of spatial orientation, such as a confusion between on, above or under the things. There does not seem to be a sufficient reason to doubt this, and to assume absence of perception of space. Due to incomplete development of speech. The late development of the relational stage of speech. The presence of a sense for orientation, which is usually present in the weak-minded, too. sets in much later.

want of understanding, but disturbance of attention. The same cause may prevent repetition, because the test sentence has already been forgotten.

Symptoms.—Aside from lingual diction, which is usually not disturbed in children, akataphasia in faulty use of the syntax will occur, which means wrong position of words and wrong grammar. Liebmann, who applies the term "agrammatism" to syntactic mistakes, differentiates three degrees:

(1) Children between two and eight years neither form nor repeat sentences spontaneously. Their sentences have the form of interjections and are ejaculated accordingly, as for instance "pussy ow!" is intended to mean "the cat has scratched me" (accompanied by gestures).

(2) The language is developed somewhat further in children equally old who speak in a paratactic arrangement of words, but without inflexions. Here the syntactic development of sentences has at least commenced, while grammar is still absent. Repeating is still partly a failure: "Pigeon up tree" instead of "the pigeon has flown up the tree." Very simple sentences, however, can be repeated. Again, the central defects are fewer and the lingual understanding is more extensive than in the first group.

(3) The last group comprises children afflicted with stammering, paraphrasia præceps and dysacusis. They spontaneously use syntactically better, though not perfect, forms, but use wrong inflexions. They are also remarkable for a tendency to peculiar word formations and inflexions, as for instance "speaked" instead of "spoke," which, however, also occurs in normal children (see p. 371). Parts of two words may also be combined into a single wrong one. These defects may persist until adult age, preferably in stutters.

The *diagnosis* is not difficult by confining the observation to the formal construction of sentences, disregarding at first disorders of speech. If there is no formal construction, simple and complicated sentences should be repeated, such as "This is a chair," "I am sitting upon a chair," etc., when syntactic and grammatical defects will become easily apparent. The diagnosis is more difficult in paraphrasia præceps and stuttering. In the former affection, slow speech is usually correct, but in the presence of both affections akataphasia is usually not discovered until after continued observation and treatment.

The *prognosis* of akataphasia is usually favorable under correct treatment, so long as the defects of intelligence are not too pronounced. The latter, however, need not be primary, but may be the consequence of lingual backwardness, in so far as the psychic progress chiefly takes place in the language, and not only in the organs of speech. In the weak-minded, the entire condition should be taken into consideration. Light degrees of disturbed speaking will, however, often persist.

The *treatment* depends upon the degree of the disorder. Special schools will not be sufficient in pronounced cases. Liebmann has devised methods of striking gestures by which to bring the designation and modifiers of a sentence more closely within the range of the child's intelligence and understanding. Thus: "The doll" (pointing it out) "is sitting" (sitting gesture) "on" (striking the seat of a chair with the palm of the hand) "that" (pointing) "chair" (taking hold of the chair). Other central defects should also be considered. If the formation of sentences is already understood, small sentences, short questions, etc., are proceeded with, as in paraphrasia præceps. The treatment requires much patience and time.

The *preventive measures* are the same as in paraphrasia. As in all disorders of speech, they consist in proper supervision of the lingual development and avoiding overtaxation of the mind.

FUNCTIONAL STAMMERING (DYSLALIA)

Stammering is the inability of uttering or correctly forming certain sounds or sound-combinations. Stammering decreases with age in children, and is in all cases present from the beginning of talking. In Germany there are 102,000 stammering children.

Causes and Origin.—Arrest of development in the motor-kinæsthetic sphere (see p. 359) must cause defects of the phonetic performances, rendering physiological stammering (see p. 372) functional. It cannot always be decided whether there is insufficiency of motor skill or of the tactile sensation of articulation. Unsuitable education and faulty speech of the surroundings also play a part, but probably only in children with an inherited tendency. So far as stammering is concerned, such is the case in 39.5 per cent. Of these 24.6 per cent. are referable to the father and 9.95 to the mother. This may also lead to defects of attention a child has to devote to his lingual performances (see p. 360). Contrary to stuttering, the cause of stammering, or disturbed production of single sound-elements, is, according to Kobrak, flaccid paresis of the lingual musculature, complicated by an atactic complaint. Higher degrees of coördination will not be formed. These children are usually backward in learning to talk and to walk. It has already been mentioned (p. 382) that sensory disturbances likewise cause stammering. Etiologically, functional stammering is to be distinguished from the organic form, although there is no difference in their outward appearance. Lispings and snufflings are more frequently caused by organic defects than any other forms of stammering and will, therefore, be discussed under peripheral arrests of development (p. 371).

Lingual arrests preponderate in frequency over those resulting from failure to overcome difficult performances physiologically.

FORMS OF FUNCTIONAL STAMMERING

(1) **Vowel-stammering** is not rare, but does not excite much attention. Initial or final vowels, or part of a diphthong, may be omitted, or an initial H may be made to precede, as in "hall" for "all." Or the initial H may be dropped, as in "at" for "hat." There are also mutilations in final sounds and diphthongs, such as occur in dialects.

The *treatment* is simple. The vowels are distinctly enunciated in precise labial positions, the child practising repetition before a mirror. If necessary, the labial position is manually corrected. Commencing a word with an aspirate, is practised by gradual transition from aspirating to whispering and vocalizing. If the voice fails to set in for an initial vowel, the child should, according to Liebmann, produce it by a coughing sound, for Liebmann counts initial vowels among the consonantal elements. Producing a hard initial sound in this way, however, may meet with therapeutic difficulties.

(2) **Stammering in Labial Sounds.**—Normally, both lips articulate together in explosives, the lower lip and upper teeth for the production of friction sounds, F and V. The explosives B and P may be confused, as in many dialects. The friction sounds F and V are frequently omitted or replaced by B and P. Using the reverse order, namely the friction sounds in place of the explosives, is less frequently observed.

The correct enunciation of the explosives is easily learned. V may be formed by slight passive parting of the lips while humming M; the labio-dental F is obtained by blowing when slightly pressing the lower lip against the upper incisors.

(3) **Stammering in Dental Sounds.**—In order to form the explosives D and T normally, the tip of the tongue is raised to the alveolar margin of the upper teeth, corresponding to the anterior portion of the hard palate. Initially, they may not be present at all, or they may be interchanged. It may also happen that the dental sounds, if absent, are replaced by gutturals, although usually the reverse is the case. Thus a boy, whose gutturals were missing, said: "dood day, Mr. Dotta," which, after practising K and G for a while, he changed into: "gook gay, Mr. Goka."

Lisping is dealt with later.

(4) **Stammering in palatal sounds (Gammacism)** is of particularly frequent occurrence. The explosive sounds K and G are normally formed between the dorsal part of the tongue and the palate. Defects are the following: Omission of these consonants at the beginning of words or replacing them by dental sounds (paragammacism).

Treatment.—The missing guttural sounds are best developed out of the letter T, which should be frequently repeated, while the tip of

the tongue is pressed downward with a spatula or a finger. In doing so, the base of the tongue is raised against the palate, where an explosive will be formed (Fig. 58).

Slight pressure behind the chin at the base of the tongue will be of assistance. If NG is missing, or replaced by G, K or N, as in "annal" for "angle," the correction is effected by separating the syllables as in "angle."

(5) **L-stammering.**—Normally, L is formed by pressing the tip of the tongue behind the upper teeth, the margins of the tongue not touch-

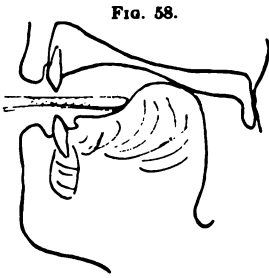


FIG. 58.
Formation of the K-sound.
A tongue-depressor prevents the tip of the tongue from assuming the T-position.

ing the teeth and the sounding air current escaping laterally. Deviations which are normal in some foreign languages are rare, but when the L-sound is formed by lowering the tip of the tongue and raising the dorsal part, a heavy sound is produced (lambdacism). The same applies to the snuffled L, which sometimes occurs in the purely functional form. On the other hand, paralambdacism, consisting in replacing L by NG, N or D, sounds ridiculous, as in "nift" for "lift," "nungu" for "Lulu."

To remove this defect, the nose is closed, the mouth widely opened, the tip of the tongue placed against the upper teeth with the aid of a mirror and a guttural is produced.

(6) **R-stammering.**—The sound R is normally produced in two ways, according to its position in the word, either by vibrating the tip of the tongue or as a uvular palate sound. Although in many ancient and modern tongues L is used vicariously for R, still, the substitution of initial L for R, as in "lub" for "rub," is ugly. Less often is D or NG substituted for R.

Treatment.—The correct lingual-R is formed by rapid alteration of dental sounds and aspiration, thus: hda, t-hda, etc. Electrical vibratory massage facilitates the formation of this sound.

(7) **Syllable and Word-stammering.**—The ordinary way of syllabic stammering consists in reversion of sounds, as described on p. 371. If entire words are changed, mutilations will result which are similar to infantile talk.

High degrees of stammering, in which, aside from vowels, nothing but a T is enunciated, is called Hottentotism (Fournier). It is very rare in mentally sound children.

Diagnosis.—All these defects of pronunciation can be found out by careful testing, designation of objects and pictures, repetition of words with one and two syllables containing the various vowels and consonants initially, medially and finally. Test words, for instance, are: Ale, blue, glance, watch, hard, rash, room, gun, tie.

By confining the tests to single sounds or syllables, some of the defects may easily be overlooked, since some stammerers can enunciate the single sounds or their alliteration, but not in combination with others.

The *differential diagnosis* would have to consider disorders of speech in paralysis, bulbar paralysis, pseudobulbar paralysis and cerebral sclerosis, if the other symptoms accompanying these affections did not exclude them. Stuttering is differentiated, aside from phonetic changes, by spasms and accompanying spastic gestures.

The *prognosis* of fundamental stammering is good in normal children, provided they do not grow up with other stammerers (stammering brothers and sisters, for instance) and there are no complications with other defects of speech.

Treatment.—When the single sounds are once formed correctly, as explained above, they should be practised in conjunction with others, forming syllables and words, so that they may be retained. Finally, independent talking should be practised. The mirror should be used for a long time to control the sound-formation, because wrong sounds are usually so firmly associated with the correct hearing picture that correction and control are impossible by the ear. Treatment always requires much time and patience. It will but rarely be possible to complete it in less than a month. Great severity will accomplish less than benevolent regard for the child's peculiarities. In this way, defects are corrected playfully and without intimidation. The little patient who suffers psychic pangs, as it is, may otherwise become so depressed as to refuse to talk. The treatment had best be carried through before school age.

Physiological stammering is no reason why talking should be carried on in that condition up to the tenth year or so, to compile children's books which they commence to get ashamed of when three or four years of age. Nurse maids with defective speech may "infect" entire families (Gutzmann). At the period of physiological stammering, there is no need for special exercises: Pure, slow, correct conversation is sufficient.

3. PERIPHERAL EXPRESSIVE ARRESTS OF LINGUAL DEVELOPMENT

(MECHANICAL DYSLALIA)

Organic stammering differs from the functional form less by the disturbance itself than by its cause, which consists in anomalies of formation of the organs of articulation, by which the lingual development is aggravated or partly arrested. Some of the lingual defects to be described, however, also occur as a functional disturbance and therefore properly belong to the previous chapter. But since mechanical causes are demonstrable in a large number of cases, it seems opportune to discuss the various forms of lisping and snuffling together.

Lisping (sigmatism) is a dental form of dyslalia which interferes with the correct formation of the friction sounds S, SS, SH, Z and ZH, and consequently also of the combinations PS, TS, X, MS, NS, etc. Sigmatism, or the wrong pronunciation of these sounds, is differentiated from parasigmatism, or their substitution, as in the other forms of stammering.

For the sake of better understanding, it will be preferable to describe the normal way of forming the sound S. Both rows of teeth are placed exactly upon each other, so that the inferior maxilla must be pushed forward, while in the normal closure of the mouth the lower incisors are located behind the upper ones. The tongue is curved upward with the exception of the tip, forming a longitudinal furrow in the median line through which the air current is conducted against the middle of the dental rows. At that place the tip of the tongue, which is depressed behind the two lower central incisors, causes friction of the air current and with it a sharp hissing sound. In forming SH, the two dental rows again stand upon each other, the lips assume a probosciform protrusion, while the tip of the tongue remains somewhat retracted behind the teeth, without touching either these or the palate.

Causes and Origin.—Lisping is the most frequent and best known defect of speech. In many cases it is hereditary, it may be purely functional by wrong position of the tongue or have an organic basis by anomalies of dentition and maxillary articulation. Considering, however, that correct hissing sounds can be produced with totally deformed maxillæ, a certain laxity of articulation must be assumed to exist, as for instance in functional dyslalia. Aside from cases where lisping occurs from the beginning of lingual development, the defect is also found to originate at the period of the second dentition. Lisping develops, preferably, when the upper or lower incisors are missing.

When I had occasion to examine 109 boys and 104 girls of the first grade, I found 25 lisping boys (23 per cent.), 18 of whom had lost their incisors, and 40 lisping girls (38 per cent.), 24 of whom had dental defects.

Young children are also apt to lisp, if eruption of the incisors is delayed. This defect may be permanent, whereas lisping due to the second dentition is usually transitory, provided that the eight incisors are completely formed.

Aside from these physiological changes, the pathological ones play an important part in dental dyslalia. If there is any disability in placing the teeth immediately upon each other owing to their forming a convex arch with closed mouth, the consequence will be a vertical anomaly or open dentition (Plate XXII, Fig. 1). If either the upper or lower maxilla unduly protrude, the sagittal distance between the front teeth is abnormally large (Plate XXII, Fig. 2); or the maxillæ may be laterally displaced (Plate XXII, Fig. 3), involving a unilateral or bilateral, inward or out-

ward dental anomaly. If there is not enough room for the teeth in the alveolar process of the superior maxilla, some of them are pushed out of the row, usually inward; or the entire dental arch is bent outward; or its margin assumes a convex or concave form. When trying to place these abnormal dental rows upon each other, they will form lateral gaps (Plate XXII, Fig. 4). Displacements may also be caused by injuries or fractures of the maxillæ, although this does not often occur in childhood. These dental anomalies do not necessarily lead to lisping, but they predispose to it in a high degree.

Coën mentions micro- and macroglossia as an etiological factor, but this does not seem to have been confirmed. Schleissner states that a so-called short or long frenulum has no influence whatever on lisping. He rightly doubts the occurrence of adhesions between the lower surface of the tongue and the oral fundus. Anomalies of the tongue itself have a very subordinate influence upon speech and cannot be held responsible for lisping.

Imitation, however, is of considerable influence. Thus, Gutzmann mentions a case, where a lisping nurse girl introduced the defect into a family, where all the children acquired it. Sigmatism is always attributable to a wrong position of the tongue which is often occasioned by anomalies of the maxillary articulation.

Lisping may assume different forms according to the position of the tongue, producing different kinds of friction sounds.

(1) **STRIDENT SIGMATISM.**—The tip of the tongue is withdrawn too far away from the teeth, and the resulting sound will resemble SH. This defect can be easily removed by positional correction of the tongue. The same term has now been given to a harsh, whistling S-sound, but this hardly ever comes under treatment.

(2) **ADDENTAL SIGMATISM.**—The tip of the tongue is pressed against the lower margin of the incisors, instead of the upper biting margin. This results in a sound similar to the English TH. A protruding lower maxilla is a predisposition.

(3) **INTERDENTAL SIGMATISM.**—The tongue is pushed between the two dental rows, which produces a friction sound against the upper incisors. It is the most frequent form of functional lisping, but will also be produced in open dentition (Plate XXII, Fig. 1), provided the tongue is not pressed upward.

(4) **LABIO-DENTAL SIGMATISM** is evidently a very rare form which I had occasion to observe in a normal girl of seven years. The lower lip is placed against the upper incisors, as in pronouncing F, while the S-position of the tongue is maintained. This produces a very sharp, almost whistling friction sound, and indeed the position of articulation resembles that assumed when whistling upon a key.

(5) **LATERAL SIGMATISM.**—The tip of the tongue is placed behind the upper dental arch, as in pronouncing L, the air current being usually pressed between the molars of one side, rarely of both sides, so that it escapes through one angle of the mouth. This defect is very unpleasant owing to the sound-association with L. SH is wrongly formed in the same manner. It is in this defect that there are usually lateral dental anomalies, forming pathological arches or lateral gaps through which the air escapes (according to Gutzmann, in 92 per cent. of the cases). Imitation is often an additional predisposing cause.

(6) **NASAL SIGMATISM.**—There is no friction sound produced between the teeth at all, the entire air escaping through the nose, and the sound produced being similar to snoring. The tongue is usually in the T-position against the upper alveolar border or against the molars; it rarely assumes the K-position with the dorsal surface against the hard palate or the velum palati. All the S-combinations, including Sh, are often, but not always, formed in this defective manner.

This defect, which is relatively rare, has been described by a number of authors under the term of "Nasal Parasigmatism," although the S-sound is not replaced by a different sound; nor is it in lateral lispings which Liebmann also enumerates among the paraforms. Gutzmann found that in many cases the function of the velum palati is impeded, due to a previously existing hyperplasia of the faucial tonsil, and attributes to this its slackness in the formation of the S-sounds. This, however, does not apply in all cases; nor do dental anomalies to which Hopmann called attention. In many cases these are purely functional disorders.

(7) **PARASIGMATISM.**—Substitution of the S-sounds by others occurs nearly always in the course of lingual development. The friction sound is usually replaced by the explosive sounds, T and D, less often by Zh, V or F. Sh is oftener replaced by Zh, while initial Sh often replaces S.

The *diagnosis* of the various forms of lispings is hardly difficult from the above description. Nasal lispings, however, which also occurs in snuffling and other disorders, must be differentiated. This can easily be done by the demonstration of correct vocalization and articulation of other sounds.

The place of air escape between the teeth can easily be ascertained by placing a finger before them, while the patient pronounces his S. According to Gutzmann, these places can also be ascertained by means of Marey's drum and the Kymographion, which enables the physician to mark the escape for every dental interspace. This graphic representation serves as a useful comparison in certain cases.

The *prognosis* is favorable in most cases, but the fact of its being so seems to be unknown to physicians and laymen alike. In some cases, however, especially in uncorrected open dentition, the difficulties are

almost insurmountable. In other cases, where lisping has been acquired during lingual development or second dentition, the defect may spontaneously disappear. This applies to addental and interdental lisping, but not to the nasal and lateral forms. The difficulties increase, if several children in one family, or even the parents, are lisping, as the treatment of a single child may be perfectly useless. The old errors are easily re-acquired, although I have known one case where a girl I had successfully treated taught her lisping sister the correct pronunciation of S. Aside from the mechanical obstacles to be overcome, the time required for the complete cure of sigmatism depends more upon the skill and diligence of the patient than on the form of the disorder.

The *treatment* consists in practising the correct position of the tongue in reference to the teeth. A dento-orthopædic correction of the anomalies of dentition is sometimes necessary as a preliminary step, but is never sufficient for a cure, as the tongue is too much accustomed to its faulty position to give it up without special practice. Nor is adenotomy a sufficient measure to correct nasal lisping, even supposing that the faucial tonsil was really responsible for the slackness of the velum. Schleisser has shown the uselessness and danger (infections and fatal hemorrhage) of severing the frenulum. A mere model enunciation of the S-sound is quite useless, as its auditory picture is too firmly associated with the wrong position of the tongue.

In practising the S-sound, tactile sensation and correction of the position of the tongue with the aid of a mirror should be used. In the first place, the incisors of both rows have to be placed on edge. By then letting the patient blow over his tongue, a broad, soft friction sound will result between the incisors, but the air current is not yet directed against the two lower, middle incisors. Several artifices may now be resorted to, in order to depress the tongue to that place, so that it may form a medial groove.

The simplest way is to let the patient blow on his finger nail, a pointed pencil or similar object. Should this prove unsuccessful, the tip of the tongue may be depressed with a thick button sound or the nickeline wire sound devised by Gutzmann. The distal end of this sound is bent downward and may be placed flat between the teeth, enabling the patient to push his tongue underneath (Fig. 59). In this way, the tip of the tongue may be fixed in the right position, the patient will have a better sensation of how it should be placed and, after some practice, he will no longer require the aid of the sound. Liebmann prefers to do without any instrumental aid at all. In lateral lisping, he presses the malar skin firmly against the lateral dental arches, so that the hissing sound can only be formed between the front teeth. Gutzmann's method has done very good service in my hands. His

instructions are to procure interdental lipping first, direct the air to the middle front and then have a correct S formed. By setting the incisors on edge, the tongue assumes at once a slightly upward concave shape. In lateral lipping, the use of sounds is indicated in most cases. Froeschels applies a prothesis made of "Stents" material. He directs the patient to bite upon it and then breaks off the part protruding beyond the front teeth. This will close up all the gaps and interspaces and he believes that this compulsory method of forming an S will achieve quicker results(?).

To remove nasal lipping, the patient's nose must at first be held closed, so that the air will be blown through the teeth. This can be rapidly effected. If the correct S-formation has once been attained, it is best to practise before a mirror combinations between vowels and consonants, as for instance with P, T, K, L, M, and N. Coën, Gutzmann, and Liebmann have compiled systematic exercises, the latter also for foreign languages. Reading and free narrations should next be prac-

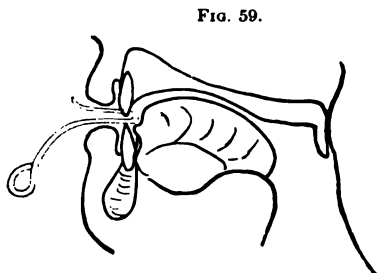


Fig. 59.

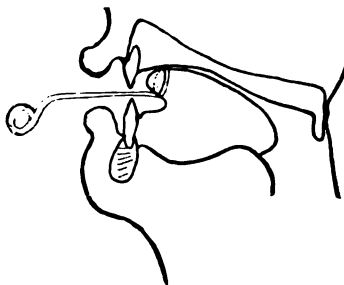


Fig. 60.

FIG. 59.—Position of the tip of the tongue for the correct pronunciation of S, with the aid of Gutzmann's sound.
FIG. 60.—Pushing the tongue backward with the ring-sound to form the Sh-sound.

tised, and not until this is satisfactorily accomplished can the patient be discharged. Sh should be practised simultaneously with S, and in lateral sigmatism it is sometimes preferable to commence with Sh. By the mere practice of a probosciform protrusion of the lips, with the teeth set on edge, the tongue will often slip back spontaneously. This should be followed by a sharp expiration. In many other cases, however, it will be necessary to push the tip of the tongue back, which is best accomplished with Gutzmann's ring-sound, which terminates in a small ring ready to engage the tongue (Fig. 60).

The time required for the treatment of the various forms of sigmatism is seldom less than two weeks, but may often extend over six or eight weeks. It is advisable to have a child cured before entering school, which can be accomplished in the fifth or sixth year. Unfortunately, however, this is often delayed until the time of marriage, probably more from ignorance than negligence.

Prophylaxis is the same as in stammering (see p. 399).

SNUFFLING (RHINOLALIA)

Snuffling is a form of nasal dyslalia in which "the nose is either open when it should be closed, or closed when it should be open" (Kussmaul). Accordingly, the affection is divided into rhinolalia aperta, rhinolalia clausa, and a combined form, rhinolalia mixta.

The physiological closure of the nasopharyngeal space during phonation has been examined by Gutzmann and confirmed by Scheier's X-ray examinations. The soft palate is raised and bent almost to a right angle about 5 mm. above the base of the uvula opposite the gyrus of Passavant, which is formed by the superior portion of the constrictor pharyngis, the musculus pterygo-pharyngeus. At that place it lies close to the faucial wall. By this arrangement the soft palate is divided into a horizontal and a vertical part (Fig. 61).

Biebedt has shown that the closing power of the velum palati is independent of age and sex, and that it is greater in the phonation of consonants than of vowels. The sound Ah requires least effort among vowels, V among consonants, while the sharp S-sound requires the greatest effort. Imperfect closure in vowels occurs only exceptionally, but M. Schmidt states that the snuffling accent will not be formed before the aperture has exceeded a space of 18 square mm. Biebedt found that the closing power is reduced in debilitated conditions (rhachitis, tuberculosis).

It is, of course, well known that M, N, and NG are the only sounds in which the velum palati allows the air current to escape through the nose.

Causes and Origin of Rhinolalia.—As will be understood from the above explanations, all defects of the velum, such as shortening and paralysis, must lead to open snuffling, and all adhesions and constrictions of the nasopharyngeal space and nose to occluded snuffling. Some of these anatomical impediments are congenital, as for instance fissures of the palate, insufficiency of the velum, and atresia of the choanæ; others develop in early childhood during the period of lingual development, such as hyperplasia of the faucial tonsil and nasal swelling; a third portion is acquired by paralysis, adhesions and injuries to the velum, etc., which, of course, have nothing to do with arrest of lingual development. As compared to these causes, a purely functional defect of the velum palati is rare, but is occasionally observed as abnormal contraction in nasal sounds in the form of occluded snuffling; or as



FIG. 61.
The velum palati in the position of respiration (black) and phonation (red). P, Passavant's gyrus; T, tubal aperture; R, faucial tonsil.

functional open snuffling, if the velum cannot be raised owing to an obstacle of long standing and the function is arrested or was never acquired; or, finally, in weak-minded individuals with defective acuity of articulation and in children of the same family with cleft palates, where the defect is the consequence of imitation. Incidental combination of open and occluded snuffling produces rhinolalia mixta. There is, besides, a purely functional defect consisting in partial snuffling which has been discussed on p. 402 as nasal sigmatism, and on p. 398 as snuffling with L, less often with P, T, and K.

Symptomatology.—Gutzmann has compiled the various forms of snuffling, on which the following explanations are based:

(1) *RHINOLALIA CLAUSA*, or closed snuffling, in which B, D, G take the place of M, N, Ng. The language sounds "dead."

(a) *Rhinolalia Clausa Anterior*.—The nasal cavities are occluded by swelling (rhinitis), hypertrophy, polyps, or atresia. Respiration through the mouth.

(b) *Rhinolalia Clausa Posterior*.—The nasopharyngeal space is partly or totally occluded by hyperplasia of the faucial tonsil, tumors or adhesions of the velum to the posterior faucial wall. Language and respiration as in (a).

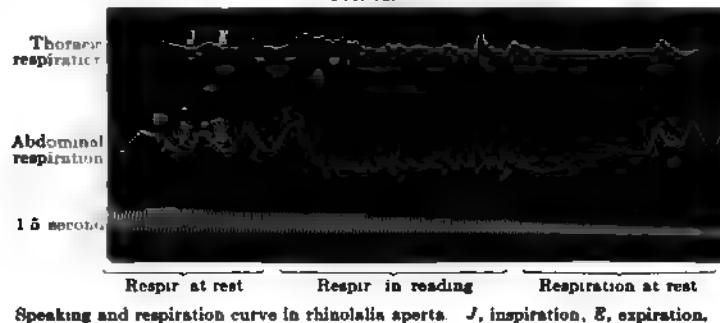
(c) *Rhinolalia Clausa Functionalis*.—The velum is contracted in the formation of the nasal sounds, M, N, and Ng, thereby causing the above disturbances of speech; otherwise its function is normal. Respiration through the nose. Abnormal contraction of the velum during respiration occurs in very rare cases and leads to the functional necessity of respiration through the mouth. The condition may also occur after adenotomy, if the previously contracted velum retains its pathological position.

(2) *RHINOLALIA APERTA*, or open snuffling.—M, N, and Ah can be spoken correctly. All the other sounds are either changed in timbre (vowels), or cannot be formed correctly or not at all (consonants). Occluded snuffling, therefore, does not interfere much with intelligibility, while the so-called cleft-palate-language of young children is almost as unintelligible as Hottentotism. Owing to the great difficulties of articulation that these children have to overcome, they learn speaking at a much later period than normal ones.

The pronunciation of those vowels suffers most where the column of articulation is relatively narrowest and the closure towards the nasopharyngeal space should be strongest, *i.e.*, in OO and EE. The nasal twang is somewhat less pronounced in O and A, but even Ah is not entirely free from it. As to consonants, N is sometimes replaced by nasal L, Ng by N. In P, T, and K the closure sound can apparently be heard distinctly, but auscultation of the larynx will at once reveal the

fact that it is neither formed by the vocal cords nor by the palatal tongue, but by laryngeal explosives, the voice setting in with a hard sound (p. 377). In K, the closure is effected by the apposition of the base of the tongue to the posterior faucial wall, thus forming a kind of vomitive sound. Substitutes for B and D are formed in a similar way and then resemble M and N. G is replaced by a bad D or N, or by the pharyngeal K. The friction sounds suffer most, or are absent altogether. F assumes the nasal snuffing sound and as such also takes the place of V. Sh and L are usually present, although with a nasal twang. L may be replaced by N. In regard to the S-formation, compare nasal sigmatism (p. 402). In palatal clefts, all the other forms of lisping are likewise present with a nasal accent, owing to the numerous dental anomalies. The lingual R is never pronounced, but replaced by the faucial R, which is formed between the base of the tongue and the posterior faucial wall. Stuttering is a rare complication. The voice is often coarse and deep owing to chronic, laryngeal catarrh. A large

FIG. 62.



portion of the inspired air escapes through the nose and is practically wasted, with the result that the frequency of respiration is greater, than normal. Rhinolalia aperta is therefore associated with increased frequency of respiration while speaking. The respiration curve is flatter (Fig. 62). Gutzmann distinguishes between the following forms:

(a) *Rhinolalia Aperta Organica*.—It occurs in congenital clefts and paralysis of the palate. (As to the former, see Zappert, vol. iv, p. 133; as to palatal clefts, see Moro, vol. iii, p. 5, and Spitzzy, vol. v, p. 23.) Congenital insufficiency of the velum palati is a rare affection, which is unquestionably caused by clefts healed in uterine life. The traces may sometimes be followed to the upper lip and palpated at the posterior end of the hard palate, where there is often a triangular segment in the median line. Uvula bifida may also be present. Although the length of the hard palate usually corresponds to the age of the child, the soft palate is shortened, changing the normal proportion of 1 : 2 to 1 : 4, according to the degree of shortening. The great distance between the velum,

with normal function, and the posterior faucial wall during phonation, decides the degree of the disturbance of speech, and the same refers to palatal clefts which have been cured by surgical interference. This condition occurs in families with hereditary palatal clefts, but I have also observed it in dwarfish cretins and idiopathically.

(b) *Rhinolalia Aperta Functionalis*.—The velum is raised in retching, etc., but does not coöperate in speaking, either because the movement was never learned, as in debilitated and imbecile individuals, or the defect has been acquired by imitation of snuffling children. Functional snuffling also occurs after adenotomy, either owing to traction of the soft velum or to want of practice of the operatively impeded velum. It also occurs from habit or auto-imitation in post-diphtheritic paralysis of the palate.

(3) RHINOLALIA MIXTA is a very important form in regard to therapy. Partial pathological occlusion of the nose or fauces, combined with patency of the velum, leads to snuffling resembling the two preceding forms, as there are both snuffling and occluded accents. This refers especially to the explosive and friction sounds.

(a) *Rhinolalia Mixta Anterior*.—Nasal hyperplasia and swelling, as for instance of the posterior end of the inferior turbinated bone; corpora cavernosa of the septum and even polyps are often observed in congenital palatal clefts. No doubt, they compensate the defects of speech to a certain extent, but at the same time lead to changes of the nasal sounds.

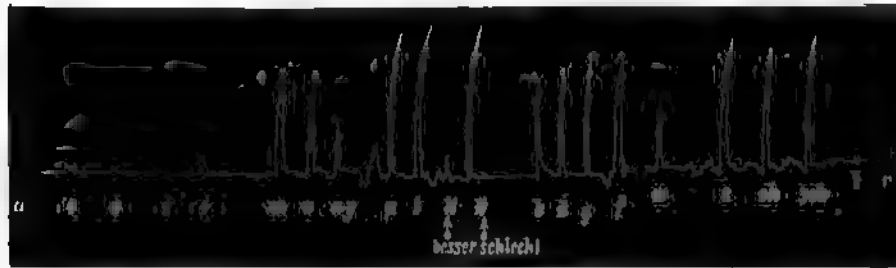
(b) *Rhinolalia Mixta Posterior*.—Hyperplasia of the faucial tonsils occludes the nasopharyngeal space and imparts to the voice a dull and doughy sound in palatal fissures, after plastic alteration of the palate or, still more, in congenital insufficiency. Hyperplastic eminences of the posterior faucial wall may become inserted in a fissure of the soft palate and effect a valvular occlusion. It is only after this has been removed (which is a technical mistake) that rhinolalia dominates the picture.

Diagnosis.—In pronounced cases of snuffling, the diagnosis will offer no difficulty, but to determine the degree and kind of impairment is not always easy, especially in rhinolalia mixta. There are special methods of examination for open snuffling, which have been compiled by Brunck. The simplest method is to keep the nose closed and open alternately, while various sounds are pronounced. If any sounds are changed except M, N, and Ng, it is a case of open snuffling. It may also be recognized by holding a mirror before the patient's nose or determining the vibration of the nose by palpation. A hearing tube introduced into one of the nostrils renders the nasal timbre more distinct to the examiner. It should, however, not be inserted into the latter's ear,

as the sonorous resonance causes earache; it should rather be held near the ear.

The respiratory movements in open snuffling have been investigated by Gutzmann by conducting the expired air through a tube into an alcohol manometer or into Marey's drum, and registering the movements of the lever on a kymographion. The normal movements of the lever in registering the nasal sounds M, N, and Ng will then serve for comparison with the pathological sounds (see Fig. 63). In order to measure the distance between the velum and the posterior faucial wall, a small instrument is used which consists of a rod, marked in centimetres and carrying a slide. The rod is placed against the posterior faucial wall, and upon phonation the soft palate will move the slide forward. On withdrawal of the instrument the distance may be read off.

FIG. 63.



Nasal curve in rhinolalia aperta (operated palatal cleft) in a seven-year-old girl. The respiratory movements are registered by Marey's drum. They should be compared with the normal movements of m and n. (Fig. 62 belongs to the present illustration.)

Occluded snuffling can usually be recognized by test words, such as "mamma," "end," "anger," "nine," etc., but it would not be permissible to assume a disturbance to be due to adenoids from these defects alone, which unfortunately is often done.

As to *diagnosis*, it is practically sufficient to distinguish the various forms of rhinolalia. In a certain sense bulbar paralysis should also be considered, but in that affection there is also genuine snuffling.

The *prognosis* is totally different, according to form and degree of the affection. It is favorable in all forms of occluded snuffling, except, of course, if there is complete adhesion of the velum to the faucial wall, and total atresia of the choanæ. In cases of open snuffling the prognosis chiefly depends upon four points, according to whether there is congenital insufficiency and whether the palatal clefts have been operated or closed by obturators, viz.:

(1) A certain amount of energy and a large amount of good will on the part of the juvenile patient are necessary to accomplish a satisfactory result in the correction of pronunciation by practising.

(2) The length of the velum—newly formed, if necessary—is of

importance. So is the quality of the obturator, which should be light, small, and soft. In infancy and childhood, however, obturators will be rarely resorted to.

(3) The prognosis is independent of the motility of the velum, a fact which will compensate for its shortness to a certain extent.

(4) The most decisive point is the behavior of the posterior faucial wall. The greater motility it shows during phonation and the more pronounced the movements of the gyrus of Passavant are, the better will the upward closure be effected. There is no doubt whatever that this musculature, especially the constrictor pharyngis, can be developed.

"The most favorable prognosis can, therefore, be made if the newly-formed velum palati reaches as far back as possible and if the motility of the velum as well as of the posterior faucial wall is as great as possible" (A. and H. Gutzmann). Rational practice will in most cases bring about either perfectly normal speech, or at least good and intelligible articulation with a slight nasal accent. Failures are rare under correct lingual treatment. Auditory control will likewise exert a certain influence. Dental anomalies aggravate the acquisition of the S-sound.

The *treatment* is (1) locally, operative or orthopædic; (2) therapy of practising. The removal of the cause in occluding snuffling belongs to the rhinological surgeon. As to adenotomy, compare Finkelstein, this Handbook, vol. iii, pp. 41-59; as to surgery of palatal clefts, see Spitzzy, vol. v. It would be in the interest of lingual development if these operations were carried out at such a period that immediately afterward practising can be commenced (at five to six years), but there are also other points to be considered. The lingual specialist should always be consulted as to the selection of an obturator, so that the instrument may not prove an obstacle to rational speaking exercises. In congenital insufficiency, Gutzmann recommends paraffin injections into the posterior faucial wall in the region of Passavant's gyrus. The loss of motility which the latter thereby undergoes is of no importance, because the velum is normally movable. Without speaking practice, the injections are of no avail.

Surgical interference with the nose and fauces in rhinolalia mixta requires the greatest caution. In case of need and the presence of an affection of the ear, Gutzmann advises that only parts of the faucial tonsils be removed. Although operation will cure the occluded snuffling, it will considerably increase the open and much uglier form of snuffling; the effect on the organs of speech will therefore be a deterioration.

Even adenotomy will not always render speaking practice superfluous, because the latter is necessary in some children to overcome occluded snuffling. In all forms of open and mixed snuffling, speaking practice is indispensably necessary.

Absent nasal sounds may be easily acquired with the aid of the

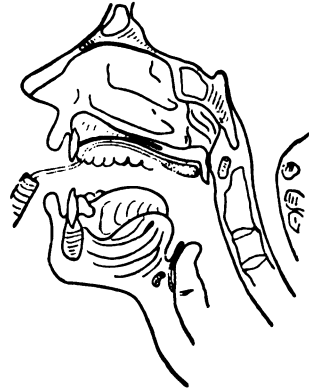
vibratory sensation, but treatment of open rhinolalia presupposes a thorough knowledge of the physiology of speech, experience and perseverance. With the assistance of the nasal hearing tube or the manometer, the patient will be able to control his own voice. A further useful instrument is Gutzmann's small hand obturator, which is made of gutta-percha, fitted to the palate and mounted on a strong nickeline wire (Fig. 64). It raises the palate, serves as a faucial obturator, as electrode and massage instrument for the velum.

While practising, it is alternately inserted and removed. The various vowels are to be sharply pronounced in a high key, with and without the obturator and passive occlusion of the nose. Correct position of the tongue and suppression of accompanying movements (grimaces) are of importance. The next practice deals with explosive sounds in conjunction with vowels, with and without occlusion of the nose. Laryngeal explosive sounds are to be avoided. Friction sounds are practised next, S offering the greatest difficulty. It will often be necessary to omit lingual R, as only the uvular "R" may be present. Formation and practising of single sounds have been discussed in the chapter on stammering (p. 396) and sigmatism (p. 400), but it will always be necessary to practise these sounds first with closed and then with open nose.

The average duration of the treatment is three months, with daily instruction. Relapses or exacerbation will hardly occur, but it will be advisable to renew practising after a time, since children will then be able to make further progress.

The other forms of mechanical dyslalia are far less frequent and important than those described. Dyslalia labialis is due to harelip and shortening of the upper lip after operation. Adhesions of the tongue do not appreciably interfere with speech.

FIG. 64.



Position of Gutzmann's hand obturator against the soft palate.

III. DISORDERS OF SPEECH

THE previous chapter dealt with lingual defects attributable to partial or entire absence of the necessary preliminary conditions, or to arrest of development in the preliminary stages. The present chapter will deal with such disorders of speech occurring in or after the period of advanced development. These are partly genuine disturbances of speech and partly symptomatic manifestations occurring in functional and organic affections of the nervous system.

1. STUTTERING (BALBUTIES)

Kussmaul calls stuttering "a spastic neurosis of coördination which interferes by pathological contractions of the vocal and consonantal closing places with the pronunciation of initial syllables, or of words in the middle of a sentence. The articulation of every sound is correct."

This definition is not entirely approved by later authors, notably Kobrak, Heilbronner, Liebmann and Otto Maas. They regard the primary spastico-paretic forms of stuttering as focal symptoms, and the primary atactic form (which in my experience vastly predominates) as neurotic. Kobrak calls them psychogenic. Troemmer believes primarily in a purely psychic cause, speaking of compulsory kinesis due to a fear of talking, of which the individual is not always conscious. As proof of his opinion he states that these individuals do not stutter when alone, but "only under circumstances, the perception of which must be mediated by association"; furthermore, that in reading, reciting poetry, whispering, singing, and speaking in foreign languages they are supposed to speak fluently. All this applies to part of the cases, although it is true that stuttering in singing is but rarely observed. The fact is that there are stutterers without any lingual impediment. Followers of Freud's theories call stuttering a hysterical fear, starting from the idea that the fear of speaking was the primary cause. Steckel mentions six (!) cases, none of which was one of genuine stuttering; there was one case of spastic aphonia and two of neurotic timidity or hysteria in adults. There remain two cases of stuttering boys, in which the disturbance sets in from sexual apprehensions at the ages of ten and twelve years, respectively, and that a cure was effected by overcoming the apprehensions. In the case of a third boy, full details are missing. Since Steckel admits that he has not yet been in a position to carry on an exact psycho-analysis of pronounced stuttering, there is no ground upon which to accept his views. Frank and Laubi have reported two cases which

are more valuable, as they point to exaggerated conceptions. However, most of the recent authors are agreed that stuttering is not a uniform affection, but can not yet unequivocally be separated into various forms, except into organic and functional.

Occurrence.—Stuttering is preëminently an affection of infancy and childhood. In most cases it occurs between the ages of two and four years, next from six to eight years, and in the period of puberty. Up to that time, then, it increases in frequency. It decreases at greater age by either being adroitly concealed or by actual disappearance. Small remnants usually remain in the shape of embarrassed expressions or accompanying gestures. The proportion between male and female is 74 : 26 in children and 9 : 1 in adults. In Germany there are 98,000 stuttering children. Gutzmann and Oltuszewsky state that the frequency of stutterers in Europe decreases from west to east, and military statistics bear this out. No race is free from it.

Stuttering may also occur as a symptom of cerebral affections and hysteria.

Causes and Origin.—Kussmaul assumes that genuine stuttering “depends upon congenital irritable debility of the syllabic apparatus of coördination.” Later investigations on the etiology of stuttering, notably by Epstein, Gutzmann, Mygind, and Uchtermann, compel the assumption that for the vast majority of cases the predisposing cause is a neuro-pathic family tendency, or a degeneration. According to Ziehen, these cases amount to 80 per cent. Leaving out of consideration alcoholism, which I do not regard as of particularly frequent occurrence in parents, also the occurrence of mental diseases (seven per cent.) cited by Mygind, idiocy, epilepsy, nervousness, neurasthenia, hysteria, deaf-mutism and asthma (seven per cent.) in stuttering families, there are numerous physical and nervous degenerative manifestations which have been definitely established in stutterers. Gutzmann states that habitual criminals furnish 3 per cent. of stutterers. He also rightly emphasizes the fact that stuttering occurs in hasty, easily excitable individuals with a labile temperament. Heredity in stuttering is also an established fact, aside from nervous predisposition.

Gutzmann found stutterers in members of the same family in 26.8 per cent., Mygind even in 42 per cent. of the cases. Mulder found among 525 stutterers 30 per cent. in whose families the affection was of frequent occurrence (see Fig. 65). Of course, an inherited taint need not necessarily lead to stuttering, unless there are additional factors, such as imitation. If, therefore, the direct hereditary influence of stuttering is to be established, none but children ought to be considered who have never heard stuttering from their parents or relatives, and, according to Gutzmann, these still furnish 8.3 per cent. (see Fig. 66).

The difference in the proportion of the sexes also points to a congenital taint. The smaller proportion of the female sex may also be due to more rapid physical and psychic development, and later to the type of respiration.

There are also a number of determining causes aside from an inherited disposition, but their value is discounted by the subjective views pervading the anamnestic statements of the parents.

Imitation of stuttering parents, brothers, sisters, and playmates is not to be underrated, although it should be unconditionally admitted that without a disposition their influence is minimal. Thus, Winckler found that 70 stuttering children had 136 younger brothers and sisters who did not stutter. On the other hand, psychic infection has been held responsible for 9.5 to 13 per cent. of the cases. The statements of parents are still less dependable in regard to the onset of stuttering after

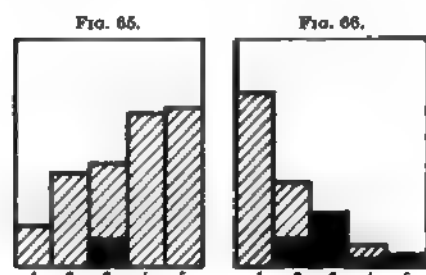


FIG. 65.—Hereditary taint. (After various authors.) 1, Coen 2, Liebmann, Troemmer and Sikorski. 3, Gutzmann (8.3 per cent. actually inherited) 4, Mulder 5, Mygind

FIG. 66. Apparent (hatched) and genuine (black) transmission of stuttering. (After Gutzmann.) 1, Brothers and sisters. 2, Father. 3, Relatives. 4, Mother 5, Grandfather.

infectious diseases (9–10 per cent.), because the influence of these affections upon the general constitution is such as to deteriorate the language. A light degree of stuttering in these conditions becomes more apparent. However, the onset of the disturbance after measles, pertussis, scarlet fever, diphtheria, parotitis, meningitis, typhoid and influenza has unquestionably been observed, while on the other hand high degrees of stuttering have been observed to disappear in severe cases of scarlet fever and to reappear

after convalescence. How unreliable parental statements are in regard to the influence of physical or psychic traumas is best illustrated by the divergence of statistical figures, Gutzmann, for instance, giving 14 per cent. and Mygind about 3 per cent.

While Gutzmann does not attach great importance to this cause, he nevertheless describes some cases of unquestionable traumatic stutters which developed after serious accidents, such as concussion of the brain. I have also known such an "unquestionable" case, in which stuttering resembled the form found in organic affections of the central nervous system and was probably a focal manifestation. However, stuttering also occurs in the form of neurotic anger on a traumatic basis, as for instance in the cases of Frank and of Laubi.

Again, stuttering is caused or exaggerated by many causes which impair the general well-being. Endogenous intestinal irritations cause reflex stuttering (Lichtinger). Other causative factors are second den-

tition, puberty and menstruation. The seasons of the year also exert an influence. In the spring and fall acute coryza impedes the functions of respiration and speech, thereby increasing the tendency to stuttering. School children suffer similarly in mid-summer from heat and fatigue; during the first school year, in which stuttering is well known to occur with unusual frequency, the demands made upon the children furnish a purely psychic factor.

Periodical stuttering with intervals of perfectly normal language, as for instance at certain seasons or in menstruation of hysterical young women, probably occurs only in individuals who have been hard stutters in childhood.

Hardness of hearing, defective organs of articulation, and adenoids, are estimated to furnish 30.9 to 50 per cent. of stutters and may be regarded as determining causes.

Up to the present, however, nearly all statistics omit to state the exact size of the faucial tonsil, and as its subjective valuation is only problematical, conclusions in this connection should be drawn with care, although it is an established fact that considerable hyperplasia of the palatal and faucial tonsils prevents a cure of stuttering by impairing respiration, hearing, and articulation.

Stuttering begins in most cases (in 245 out of 466 cases of Mulder) before the lingual development is completed, and again between the ages of six and eight years. The difficulties caused by the disproportion between lingual understanding and ability to speak (see p. 362) and the efforts to overcome the same may arrest speech in the attempt of combining syllables. The depression caused by the delay in the motor discharge of conceptions and by unsuccessful attempts at speaking may not only lead to dumbness, but also to serious spastic manifestations.

Bloch attributes his "oral stuttering" direct to a mechanical impediment to articulation.

Furthermore, the natural ataxia of a child going through the first evolutions of speaking is increased by the predisposing factors referred to, disturbing more than normally the correct lingual coördinations and often persisting for life.

Kobrak is well justified in looking upon the occurrence of spasms as a failure of the levator musculature necessary for the formation of voice and sounds, and contradistinguishes them as spastic paresis of the levators from the flaccid paresis of the constrictors in central stuttering. Both forms of paralysis produce in turn secondary atactic manifestations which find expression in exaggerated uncertainty of forming sentences in accordance with the degree of the excitement. This would explain why auditory mutes, stammering or spluttering children later

acquire stuttering. These are primary atactic stutterers, whose spasms develop in the course of time and become exaggerated under psychic influences. On the other hand, Kobrak attributes the spasms of primary paretic stutterers to hypothetic focal affections. The former are increased under excitement; the secondary atactic phenomena become less prominent, and this form of stuttering is much more uniform. The alternation between the periods of almost correct speech and stuttering, and the diminution of the manifestations under the influence of greater attention, however, are characteristic signs of the primary atactic forms. Kobrak distinctly admits that the two groups cannot be sharply separated and that there are mixed forms. His explanations of the etiology and pathogenesis of stuttering may well be accepted, with the proviso that primary paretic cases are very rare.

Of whatever etiology this disturbance of coördination may be, it is complicated in a great number of patients by psychic factors which exacerbate the complaint. There are expectancy and anxiety. The fact of speaking being interrupted is not necessarily felt by the children as a disturbance, but unfortunately their attention is nearly always drawn to it. No wonder, therefore, that an "expectant neurosis" (Kraepelin) will develop. "The distressing anticipations," which according to Kraepelin precede an unpleasant event owing to a "gradually increasing internal tension," are partly expressed by "all kinds of motor impulses," and partly impair "the certainty of action." "The pathological development (of expectant neurosis) is brought about by the fact that the distressing disturbances do not manifest themselves on one solitary occasion, but repeat themselves with regular daily occurrences. This gives rise to a continually progressing expectant angor which may reach an exceedingly high degree and persistently dominate the entire mode of life." In children this is chiefly caused by wrong treatment on the part of those about him (exhortation, scoffing, punishment), in consequence of which they purposely increase their dynamic accent. This in turn leads to increased stuttering and finally to a fear of speaking due to positive artificial incitation. The fact should be emphasized, however, that psychic depressions and arrests are not always associated with stuttering. On the contrary, they are rare before the seventh year. This is not only borne out by the conduct of numerous children, but also by the fact that older children and adults often distinctly repudiate any expectant tension, and even more so the fear of talking; they cannot even understand why "such a fuss" should be made over their impediment, as they themselves are not disturbed by it.

Gutzmann's clinical division of stutterers into three groups practically corresponds to the etiological and pathogenetic differences and may therefore find a place here.

GROUP I.—Distraction and inattention are characteristics. Repetition of badly spoken sentences will bring about an improvement or an apparently normal result. Speaking is better in school and in the presence of the physician, than at home and with playmates. The majority of children belong to this group and principally exhibit primary atactic forms.

GROUP II.—Increased attention deteriorates speech. "The volitional attention changes to irritation, which increases the spasm." This group comprises more children who have acquired stuttering at a late period than those who commenced stuttering during the development of speech. There are primary paretic forms amongst them.

GROUP III.—Secondary manifestations of stuttering, expectancy, depression, angor, occupy the foreground and dominate the picture. These grave forms are very rare in childhood.

Aphthongia, consisting in "a spasm confined to the hypoglossus region which does not occur except when a person is just about to speak" (Gutzmann), is wrongly differentiated from stuttering. Sudden failure to speak without spasms occurs less often; according to v. Sarbo it is a mono-symptomatic hysteria.

Symptomatology.—The disturbances of coördination in stuttering have been principally investigated by Gutzmann with the aid of physiological technique. They manifest themselves in spasms of all muscular regions of respiration, phonation, and articulation, which are necessary for all forms of speaking, also in associated movements and uncertainty of formal speaking. There are also a number of degenerative signs, nervous and psychic symptoms.

(1) Respiration while speaking, even when not stuttering, deviates in various respects from the normal (see p. 377), which is not the case at rest. Observation and pneumographic examination reveal the following respiratory disturbances, which are partly primary and partly secondary.

(a) The normal anachronism of respiration, while speaking, is usually absent, the thoracic and abdominal curves being synchronous, or nearly so.

(b) Inspiration interrupts the flow of speech much more frequently, being superficial. The vocal cords do not open to their maximal extent, or even approach each other a little, owing to a functional paresis of the postici (?), producing a hard friction sound from perverse action of the vocal cords.

(c) Some stutterers are in the habit of forcibly expiring before commencing to speak, although there is practically no respiratory air left to enable them to speak. This may displace the entire speaking part of a pneumographic picture as compared to the part showing respiration at rest. This advance respiration is so striking as to be observed without the examination referred to, and leads to considerable disturbance of speech (Plate XXIII, Fig. 1).

(d) This is still more evident in tonic and clonic spasms, which are usually more pronounced at the diaphragmatic than the costal movements. Thus, if they occur only in the abdominal curve, they may disappear in the costal one, while speaking proceeds without interruption, although the diaphragm stands still in the position of inspiration or expiration. In clonic spasms, expiration is continually interrupted by slight inspiratory movements (Plate XXII, Figs. 1-5).

(e) The spasms may of course continually change in the various muscular regions, disturbing the uniform character of the thoracic and abdominal movements. Instead, there will be opposite movements, shown by a rise of the abdominal and a fall of the thoracic curve, or *vice versa* (Plate XXIII, Fig. 6). When speaking is done with, respiration often stands still for a time, before the regular rest respiration resumes its function—usually with a sigh. This is also observed in nervous children who do not stutter.

All these respiratory disturbances may be primary without accompanying spasms of the vocal and articulating musculatures. If, however, spasms in the upper air passages are to be overcome, they will occur secondarily. The closed vocal apparatus is forced open by explosions of speech, or, failing this, a burst of inspiration will do so, with the result that the stutterer will commence speaking with an inspiration. The respiratory spasms may also be preceded by spastic movements of the entire larynx without closure of the glottis, also of the tongue and lips; the contrary may also happen, when the spasms are caused by the respiratory disturbances.

(2) The movements of the larynx very often deviate from the normal, but not as uniformly as the respiratory movements. Aside from the inspiratory way of speaking above referred to, and from the hard initial sound which unfortunately is present in many normal children, there are also tonic and clonic spasms of the larynx, causing vowel-stuttering owing to the spastically enforced closure of the glottis.

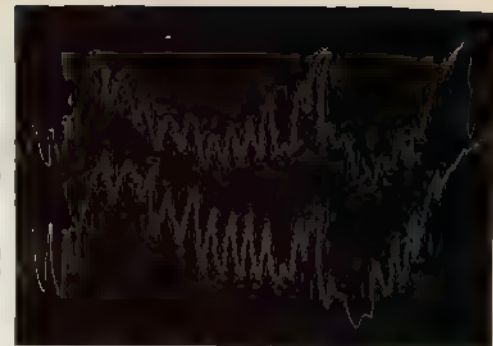
They can but rarely be observed by the laryngeal mirror. The latter reveals first the spasms of the glottis musculature (first described by Gutzmann) involving, according to Oltuszewski, the levator and constricting muscles. The vocal cords and ventricular bands are often pressed so firmly together that the latter completely obscure the former. In other forms the vocal cords are not close together in spite of continued spastic conditions, leaving a narrow fissure between them. Furthermore, there are twitching or intending movements, which cause their repeated closure previous to actual phonation. On the other hand, the opening movements may predominate, leading to a picture similar to recurrent paralysis. Thus, there will be complete or intermittent vocal stuttering according to the kind of prevailing spasm. Strong pressure

PLATE XXIII.



Respiration at rest Inspiratory spasm Inspiratory spasm of thoracic breathing Advance respiration in abdominal breathing

FIG. 1



Resp at rest Speaking in position of inspir. Rest Inspiratory retention spasms to produce D

FIG. 4



Respiration at rest Expiratory spasm Rest Expiration interrupted by slight inspiratory movements on attempting to speak

FIG. 2



Rest Firm posn. of diaphragm in position of inspiration Incomplete movement of thoracic and abdominal respiration Rest

FIG. 5 (not 5) (Transposed by the printer on account of space)



Respiration at rest Expiratory spasm Inspiratory spasm

FIG. 3



Respiration at rest Clonus spasms of the diaphragm in position of inspiration

FIG. 5

FIGS. 1 TO 5. Speaking curves of stutters.
(To be compared with the normal curves of FIGS. 50 and 54)

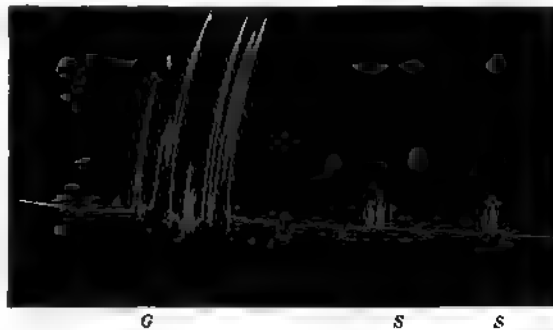
movements as well as wrong phonation (see p. 473) will cause secondary injury to the larynx, which can be recognized by small erosions of the vocal process, reddening and swelling of the vocal cords, which also show small eminences (singers' nodules). A continued falsetto voice is also often met with. Local affections of the larynx are again productive of spasms owing to the effort required in overcoming the obstacles to phonation they create. Abnormal movements of the larynx which are externally visible have already been mentioned.

(3) The various spasms of articulation are accessible to direct observation, except clonic spasms of the velum palati. The soft explosive sounds B, D, G, in which the closure must be actively opened, cause greater difficulties to the stutterer than P, T, K, in which passive opening occurs by the air current. Besides, the opening movement in soft explosives (*mediæ*) requires a more delicate coördination than in the hard ones (*tenues*).

Closure spasms of the lips occur in labial sounds, of the cremaster—less often of the tongue—in dental and palatal sounds. The opening movements are either almost normal with exaggerated acuity, or inspiratory, or they occur at the end of expiration after the palate has closed and the air has escaped through the nose. Clonic and tonic spasms of the labial musculature can be seen, those of the cremaster and tongue can be felt at the cheeks and fundus of the mouth. Clonic spasms, especially snap movements of the inferior maxilla, also occur with other sounds (see below). They cause intermittent closure of the velum, as can be seen from a reproduction of the nasal air gusts (Fig. 67). Friction sounds are spastically overextended and often uttered without phonation, as for instance *f* instead of *v*. The velum may fail to close, with the consequence that the air escapes through the nose with a snuffling noise. Spasms of the fundus of the mouth and velum also occur with L, rarely with R. In vocal *mediæ* and friction sounds the spasms may be confined to the larynx, in which case the sounds set in with a distinctly audible noise of the glottis.

The numerous peculiarities of disturbed coördination of the various muscle groups are not exhausted, but their principal manifestations have been described in the above. They manifest themselves by interruption of speech and exaggeration of the dynamic accent. Kobrak summarizes

FIG. 67.



Curve of air gusts, escaping through the nose in clonic spasms of the velum palati (in pronouncing *G* and *S*).

them as insufficiency of the levators in reference to the constrictors of all muscle groups serving the purposes of speech. Liebmann regards intentional exaggeration of the consonantal elements (explosive sounds, including closure of the glottis) as the primary cause, and respiratory disturbances as secondary. This view is contradicted by the pneumographic investigations of Gutzmann and his disciples, who found pathological respiration even in the fluent talk of stutterers. Spasms of respiration are undoubtedly the primary manifestation of coördination neurosis in the majority of cases, although secondary spasms of the apparatus of articulation may also occur. In persons who stutter without having any lingual impediment (*forme frustes*) the respiratory disturbances can only be established by pneumographic examination.

(4) Occurrence and intensity of the spasms are also dependent upon the manner of phonation, manner of talking, and impressions. It has long been known that they disappear partly or entirely in whispering. Gutzmann states that about one-third of all stutterers experience no impediment when whispering. This is explained by absence of the hard initial sound and reduction of the dynamic, possibly also the musical, accent of speech. The relief given by aspirated initial sounds causes some of these children to resort to voluntary whispering (p. 391). Talking with a soft voice in a deep key also improves the disturbances owing to the entirely new coördination they produce. The manner of phonation and rhythmical expression following the change of accents in monotonous speech, and the same causes combined with the pleasurable sensation in singing, diminish in most cases the occurrence of spasms, but there are also singing stutterers. The same refers to reciting. Reciting, slow speaking and reading are easier to the atactic stutterer, because he has the advantage of a given form which, in spontaneous speech, he is compelled to find for himself. Reproduction offers less difficulties than production, because more attention can be given to the requirements of speech. Even those who stutter hard from increased attentive tension can repeat and read with greater facility. On the other hand, there are cases in which spontaneous talking can be accomplished almost fluently, while reading or repeating is considerably disturbed. Gutzmann attributes this to a sensation of displeasure in being bound to given words, and to fear of so-called "difficult sounds." The ability displayed by hard stutterers in talking fluently in playing actor in children's theatrical performances, and in imitating peculiarities of other people's voices, is best explained by the changed timbre and deflection of attention from the act of speaking, in which the prevailing hilarity coöperates. These are cases belonging to the second and perhaps third groups. Angor and ire may arrest speech as well as promote it, and this applies with particular force to hard stutterers. Secondary sensations caused

by the act of speaking itself exert, after all, the same influence as accidental primary sensations of angor and ire. This explains why the relief experienced on overcoming the first difficulties enables children to continue, while "in other cases the negative sensations caused by the failure to do so may be increased to such a pitch of intensity that further speech is absolutely impossible." Gutzmann designates this as voluntary silence as an expression of arrest of the will power, caused by the sensation of displeasure.

The reciprocal effects between impression and lingual accents, or rhythm, dominate the occurrence and intensity of stuttering in all the forms described.

Aside from lingual stuttering, there is also writing, walking, piano, violin and trumpet stuttering. In children none but the first form occurs occasionally.

Accompanying gestures in stuttering are defined by Gutzmann as "abnormal movements occurring in voluntary movements of muscles which are not coördinated to the intended movement." Part of them, however, are voluntary, as are certain anomalies of speech which are intended to help or cheat away over-difficult sounds. They might be called "evasive movements." Gutzmann divides the same into primary and secondary. The first group includes all those cases where irradiation of volitional impulses occurs in the muscular region adjacent to the organs of speech. This would refer to grimaces, as for instance distortion of the mouth in labial sounds (Fig. 68), to spasms of the external laryngeal muscles in phonation, and the spastic movements of the accessory respiratory muscles in spasms of the diaphragm.

Secondary movements are at first purposely used in order to facilitate speech, but later become stereotyped manifestations without which speaking becomes impossible. They are bound to accompany speaking without, however, improving it, and it is only in this respect that they are sometimes distinguished from the explanatory gestures made by normal individuals.

Children, as a rule, avail themselves of easily recognizable, striking gestures. They will swallow, shake their heads, strain their cervical muscles, bend their heads, twitch their eyes (Fig. 69), clap their hands, pat their legs or back, strike a chair or the wall they lean against. They will push and strike at objects, stamp their feet, and execute peculiar walking movements, etc. A boy I had observed crossed the hands behind his back and struck at the elbow with his fist; another one half-raised himself in an armchair, rocking his body while talking, with his arms resting on the chair. Sometimes a hand is repeatedly put to the mouth as if to conceal the spasm or wipe it away. This is rather an infantile movement of embarrassment. Accessory words are sometimes

used to overcome the difficulty, such as *well, yes, hm, and then, or* sounds are purposely avoided and replaced by others. Initial *t* may be overcome by preceding them with the aspirate, such as *E* for *Anna*. Consonants are less often replaced, as in *B* instead of *K* instead of *G*. This has an outward resemblance to stammer although the correct sound may really be possible of formation. may lead to a compulsory peculiarity of expression, if certain words avoided owing to their difficulty and replaced by others of an inadequate meaning.

(6) Combinations of stuttering with other lingual defects are frequent, for the reason that stammerers, splutterers, agrammatists,

FIG. 68



FIG. 69



FIG. 68—Spasm on pronouncing *B* overcome by primary accompanying movement—distortion of the eyes.
FIG. 69—Spasm on pronouncing *D*. Secondary accompanying movement—twitching of the eyes.

tering. Careful examination of stutterers reveals the fact that in almost all cases there is uncertainty of grammatical and syntactic diction, amounting to traces of agrammatism and akataphasia, which often leads to gross errors of expression.

Uncertainty of diction leads to constant confusions in grammar and syntax, the recognition of which again disturbs the flows of speech. Older boys are apt to mix up two phrases of similar meaning, thereby forming inadmissible combinations.

(7) Neuropathic signs which are mostly hereditary (see Thiemich, this Handbook, vol. iv, p. 295) are present in a great number of stutterers; so are physical signs of degeneration. The principal ones, so far as they have been connected with lingual defects, may here be mentioned.

There is nearly always abnormal excitability along with pathological fatigue in performing physical or mental work. There is also great distraction, especially in children afflicted with stuttering of the atactic type. Von Sarbo has written on the relationship between disorders of speech and *maladie des tics*, and Bonnet on their relation to stutterers.

Both affections have many points in common, as for instance similar and dissimilar heredity and determining causes, but the tics never occur as early as stuttering and they attack both sexes. They also resemble each other in symptomatology, as for instance in the evasive movements which Bonnet designates as "antagonistic ruses of war," and in the secondary symptoms. Upon close observation, more or less pronounced tics occur in many stuttering children, especially of the facial musculature when not speaking. According to my experience, the tics improve with the removal of the disorders of speech, provided that the children are correctly treated from a psychic point of view.

Gutzmann states that masturbation is often practised by stuttering boys; there is nothing known about girls. Liebmann is inclined to regard it as rather important. Steckel, who has rediscovered this fact, believes that masturbation is of the greatest etiological importance, and as proof for his assertion mentions—one case. Considering, however, that masturbation is very widely practised and that its injurious effect is an uncertain factor, no great attention should be paid to it. As to treatment, see Thiemich, p. 364. Gutzmann also states that enuresis nocturna often occurs in stutterers, and Mygind estimates the cases at 2 per cent. (see also Thiemich, p. 374.) There are also local perspiration, dilatation of the pupils while stuttering, and other signs. Gutzmann also calls attention to numerous physical signs of degeneration, such as deformities of the palate, syndactylism, cryptorchidism, supernumerary vortices of cranial hair, and pronounced hemihypoplasia of the face (5 per cent. of the cases). Unequal innervation of the tongue and mimic

musculature have been described by Otto Maas. Deviation of the protruded tongue is said to occur twice as often in stuttering as in other children (Fig. 70). It should also be mentioned that many of these children are afflicted with manifestations of neurolymphatic diathesis (Pfaundler, vol. ii, p. 233). This "readiness for disease" occurs in twice as many boys as girls.

Other complications of stuttering mentioned by Gutzmann are Basedow's disease and epilepsy, without, however, suggesting any relationship between them.

The *anamnesis* cannot be exact enough in the interest of treatment. Neuropathic symptoms, diet (preference for meat, coffee, etc.), digestion, sleep (Kraepelin's type of nervous sleep), idiosyncrasies, bad habits (chewing the nails), temperament and character, should be carefully inquired into. Statements about origin and duration of stuttering are not always dependable; it is far more important to find out disorders of speech and nervous or psychic affections in the family.



FIG. 70.
Hemihypoplasia of the face, the tongue deviating to the right. (There is also arrest of development on the right side of the middle ear.)

The *diagnosis* of stuttering offers no difficulties so long as there is an opportunity of watching the impediment and the exaggeration of the dynamic accent. This is often impossible, because children are apt to speak without stuttering during the medical examination. A careful examination, which indeed is necessary in all cases, should then establish the presence or absence of the above symptoms. Spasms of the respiratory musculature can often be discovered when children talk undressed. The pneumographic examination will reveal further details in regard to polypnoea, synchronism of the thoracic and abdominal respiration, etc. Faulty inspiration (perverse action of the vocal cords) can easily be perceived by the ear. Weakness of the respiratory musculature is present if children cannot hold their breath for a few seconds and cannot expire slowly for ten to fifteen seconds. Spasms of the larynx can be established by auscultation; they can be seen and felt in the region of the muscles of articulation. The various forms of speech should be examined into, consisting in spontaneous speech, answers, repeating, reading, and perhaps reciting, singing, and whispering. The rate of speech, the pitch and strength of the voice should be noted. Accompanying movements, accessory words, and tics, can hardly be overlooked. A general physical examination is also of great importance.

Differential Diagnosis.—Stammering might be considered in psychically depressed children, but stammering distinctly differs from

stuttering by a general kind of fluency, uninterrupted by incoördinated movements. Stammering is a mistake in the pronunciation of certain sounds. But both defects may occur together, and stammering which is never absent may be the only apparent defect at the examination, while stuttering may be absent for the time being. These cases require prolonged observation. The differentiation between spluttering and stuttering may be more difficult if a splutterer in his hasty talk becomes so confused as to be prevented from finishing his sentence. But there are no spasms. Should the latter occur, the diagnosis of stuttering is safe. A combination of both forms cannot always be recognized at the first examination, but the presence of spasms and of pararthric disturbances, distortions and mutilations of syllables, etc., is proof of the complication with spluttering.

Hysterical stuttering, which in its outward appearance is not distinguished from the genuine form, is to be considered in older children. Its mode of development together with other hysterical signs will point the way (see p. 438). Disorders of speech resembling stuttering occur in chorea, progressive paralysis, and multiple sclerosis. Stopping between syllables in the last-named disease can only lead to errors, if other symptoms are absent. The respiration curve, however, shows no spasms. It has already been mentioned that so-called aphthongia does not really differ from stuttering.

The *prognosis* of stuttering is one of the most difficult problems in this affection, the most experienced being liable to the widest errors. In early childhood it is considered more favorable, because the disturbance of coördination has not been of long standing and a young child will easily unlearn the habit (see p. 376). The somatic and psychic constitution is of importance. In underfed, wrongly fed, anæmic, and sick children (epileptics) the prognosis is bad, especially if for external reasons (poverty) nothing can be done for them. The same applies to seriously degenerated children, their energy being usually insufficient to support our therapeutic measures. Intelligence is a favorable factor, inasmuch as intelligent children have a greater liking for learning, but on the other hand less sprightly children do not observe the defect as much and are not so easily subject to linguophobia. The intelligence of the persons about the children is far more important, and this is often sadly wanting. The pedagogic mistakes which are here committed are almost incredible. There are not only exhortation, scolding, and corporal punishment, but even practising of the most astounding "difficult words," until the child gets thoroughly frightened and stutters more than ever. The poorer classes do not trouble at all about the training, and the better ones leave it to the servants, which amounts to the same thing. Over- or under-training renders the prognosis less favorable,

particularly if there is no uniformity of treatment. The same applies to school pedagogics, although generally more sense prevails here. The confidence and attachment of a stuttering child to the physician and teacher are favorable factors in the prognosis. For the same reason there is hardly any chance of success if the influence of the surroundings is detrimental.

The *etiology* of any particular case of stuttering is of importance for the prognosis. According to Gutzmann, children can be more easily cured who commenced stuttering during the development of speech than those who have acquired the affection later by imitation. If the disturbance sets in after acute diseases or at the time of puberty, and is treated early, the prognosis is more favorable than in cases of long standing.

Among the various forms of stuttering, the one due to vocal disturbances gives the worst prognosis, the one due purely to respiratory irregularities gives the best. The prognosis is less favorable in children who are psychically depressed or very distracted. But any apprehensions in this respect should be withheld from the patients. It is also an unfavorable sign if the change of timbre and whispering does not exert any influence upon the lingual disturbance.

In all cases, however, the prognosis can only be established after carefully pondering the results of the examination and after prolonged observation. Caution demands reticence for the time being.

Spontaneous cures will occur. They are rare in boys and demand unusual energy, while in girls they occur more frequently during the period of physical development.

Unsuccessful attempts to effect a cure render the prognosis less favorable. These may be due to the impatience of parents who discontinue the instructions too soon, but more particularly to the interference of quacks who promise the most splendid results and institute measures the uselessness and danger of which are unfortunately not always known even to physicians.

The *treatment* of stuttering is one of the most difficult tasks in the cure of lingual disturbances and cannot be successfully undertaken except by drawing upon the resources of the entire medical art. Without medical and pedagogic treatment which takes into account all the nervous and somatic defects and ailments of stuttering children, the mere therapy of practising is often powerless.

Kobrak (as well as Heilbronner) has laid down some general points on the therapy of stutterers, based upon his views on the etiology and pathogenesis. "Psychic arrest should receive psychic treatment (sanatoria for children); deficient intelligence, which often consists merely in want of attention and concentration, should be improved by suitable

pedagogic treatment. Defects of the centres of speech require practising; arrests localized in the peripheral organs of speech may require operative interference." This, however, is not intended to be a hard-and-fast rule. "The treatment has not the significance of instruction, but is a therapeutic task for the physician" (Heilbronner). Where correct treatment cannot be carried out owing to the surroundings, the views or capabilities of the parents, the child should be placed in an institute. The disadvantage of so doing is the seclusion and uniformity of life which deprives the stuttering child of an opportunity to exercise his lingual dexterity, the more so as he is kept away from all psychic emotions. On the other hand, the necessary pedagogic and dietetic measures can be better observed in an institute. Recently, Gutzmann has again referred to the importance of this point, although it was well known to our remote predecessors, but disregarded in the nineteenth century. The treatment of nervousness, anæmia, constipation, etc., cannot be entered into in detail at this place. More detailed information on this point has been supplied by Schlossmann and Sommerfeld in vol. i and by Pfaundler in vol. ii of this Handbook. It may, however, be mentioned that undernourished stutterers of the poorer classes should receive an invigorating diet, supported, if necessary, by medication (iron, leivico water, cod-liver oil), while wrongly fed, pale, and lean children of the better classes should receive a bland, non-irritating diet. The latter class of patients, who are usually overfed with meat and eggs, and possibly receive alcohol as a tonic, usually suffer from chronic constipation, and will not only do very well on a vegetable diet, but may even spontaneously lose their lingual disturbance, while practising alone would have scarcely any effect.

Chronic constipation should, of course, be removed by suitable diet and gymnastics.

Sensible parents are soon convinced of the usefulness of this plan and carry out a proper diet. A boy I had treated eight years ago and who is subject to periodical slight relapses is given this diet on each such occasion, after which speech is at once corrected to the satisfaction of the parents.

Stuttering is no indication for the removal of the tonsils or for nasal operations. Should such become necessary, it had better be done under anæsthesia ("twilight" ether sleep; Spitzzy, vol. v, p. 5) or under careful local anæsthesia after Ruprecht. Nearly all authors have observed that adenotomy may effect considerable improvement in mouth-breathers, but it is a rare occurrence.

I have observed only once the "awakening" of a nine-year-old backward girl after this operation, which was followed by rapid disappearance of the lingual defect after a few lessons had been given in practising.

Mild hydropathic measures are recommended by Gutzmann as a general treatment of the underlying affection, consisting of friction with tepid water or at room temperature, long baths, but no cold baths or douches. Pure air, curative treatment, if possible in vacation colonies or forest schools, are beneficial. As to medication, the bromides are seldom indicated in children. I like to prescribe *infusum valerianæ* with good symptomatic results, which help three- or four-year-old stutterers over their difficulties and therefore have a curative value. The type of sleep of these children requires insisting upon sufficiently long hours, going to bed in good time, and rest before meals in school children, if they require such. Masturbation has been discussed by Thiemich in this Handbook, *l. c.* Electricity has, according to Gutzmann, none but suggestive value. He rightly regards hypnotism as quite objectionable in children.

If, to take a case mentioned by Gutzmann, a girl of eleven is hypnotized several times a week for a stretch of four years, exacerbation of the trouble cannot cause surprise. I had to treat a boy of eleven who had been hypnotized every other day when seven years old, and once a week at the age of eight, although his lingual defect grew constantly worse. He was a typically distracted stutterer (primary atactic form) with tics, and who nevertheless yielded to sensible therapy without any particular difficulties. Troemmer, who is an advocate of hypnotism, reported only adult cases. Steiner used hypnotism in conjunction with practice exercises and found that hypnotism had no influence on the result, which was the same as in non-hypnotized cases. There was the same percentage of cures.

On the other hand, sensible psychotherapy which takes into account the etiology of the case, the psychic life and temperament, is of inestimable value. Psychic training is to be preferred to direct suggestion. Stuttering children should be met with great calmness and indefatigable patience in view of their uncertainty and fright, so that they may gain confidence in the physician and in themselves. Parents and teachers alike must display an unshakably even temper. Should there be difficulties in the way in this respect, the child should be removed from school or home for months. No doubt there are many stuttering children who have been badly or wrongly brought up and whose lingual defect will be improved or suppressed by increased attention. These distracted patients should, according to Gutzmann, be treated with calm severity, but not with unfriendliness or scolding. Severity, wrongly applied, may do more harm than too much kindness. Liebmann indeed comes to the extreme conclusion that in the treatment of stutterers it is less a question of the method employed than of the personality of the physician. (Neumann, vol. i, p. 254.)

The object of the special treatment is to practise physiologically correct speaking. Gutzmann states particularly that this should be done on physiological principles and that consequently "Gutzmann's Method" should not be spoken of as a cut-and-dried measure.

The curative plan worked out by A. Gutzmann has, of course, to be adapted to each individual case. His principles are the following: "The movements necessary in speaking must be consciously and physiologically practised, suppressing all accompanying movements. The act of speaking should then take place unconsciously without any par-

FIG. 71.



FIG. 72.



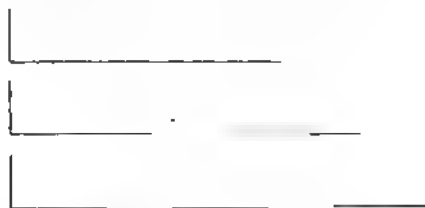
FIG. 71 —Correct breathing exercise. Deep inspiration without forced movements.
FIG. 72 —Wrong breathing exercise. Inspiratory raising of the shoulders, straining of the cervical muscles, and retraction of the abdomen.

ticular attention being paid to it." Respiration, phonation, and articulation are carefully practised just as they are used in speaking.

(1) The object of the respiratory exercises is to accustom the child to correct breathing for speaking purposes, consisting in deep, short, noiseless inspiration, not pumping the lungs full to their utmost extent, and very slow, soft expiration. Audible, snapping inspiration (perverse action of the vocal cords), accompanying movements like raising the shoulders, straining the abdominal muscles, raising the tongue and cutting grimaces are to be avoided (Figs. 71 and 72).

The respiratory exercises may be combined with gymnastics. Inter-

pulation of pauses in expiration, which are not to be interrupted by slight inspirations, is practised to get accustomed to the pauses indicated in speaking (Fig. 73). The respiratory exercises are controlled by the child placing his hands against the sides of the thorax and observing himself before a mirror. It is often advisable to have the exercises gone through with the upper part of the body undressed, so that wrong movements may be the easier observed. Pneumographic pictures taken from time to time will demonstrate any progress made. The following diagram will illustrate the scheme of respiration; the vertical line indicates inspiration and the horizontal one expiration.



(2) In order to fuse a complicated coördination, such as onset of voice and initial vowel, it is advisable to practise the various components of pho-



Wrong respiratory vocal exercise ha-a-, interrupted by a slight inspiration in the middle J, inspiration, E, expiration

nation separately and then to practise their fusion so as to build up the movements necessary for coördination. This applies particularly to vocal stuttering. Thus, from aspirating with an open glottis we pass on to whispering with a closed vocal, and open respiratory, glottis (see p. 377), and finally to the actual onset of the voice in various vowel positions. Later, transition is practised direct from whispering or aspiration to the actual voice. Next comes the soft initial sound which is more difficult to produce by the stutterer than the hard one. The voice should be pitched low, as in all exercises; the

intensity of the voice may be very slight at first. The vocal exercises are combined with the above respiratory exercises. For instance:

h	_____	a	_____	same with o, oo, ay, ee.
	Breath	Whispering	With voice	
h	Breath or	a	_____	
	Whispering			
ha	Pause	a	_____	
	Whispering			
wrong:—				
ha		a	_____	

(Compare Fig. 73.)

(3) Exercises in Articulation: Consonants, too, may have to be practised before the mirror, first without uttering a sound, then as a whisper, and finally in conjunction with respiration and voice practice.

The explosive sounds should be followed by an aspirate, and the persistent sounds (which are usually friction sounds) are practised first without and then with voice, and stretched. For instance:

p	ha	
f	ha	
v	a	

The length and kind of exercise depend upon each individual case. If no difficulties are encountered, which is often the case, it will be useful to pass on to words and small sentences with correct respiration and long vowels. They are first spoken in a monotonous tone, following each other monodynamically without interruption. The intention, however, is not to teach artificial speech, devoid of all musical and dynamic accent, and it is necessary, therefore, to adopt the natural rhythm at an early date without, however, relinquishing the other exercises. Reading exercises, repeating stories in short, simple sentences, answering questions, free narration of events, and conversation will bring the child step by step to fluent, formal, and technically correct language. The best and quickest way to achieve this result depends upon each case, especially upon the etiological factors. Practising, however, not only brings about physiologically correct speaking, but also directs expectation away from the conception of sounds to the technique of the language and to the parts already learned by practising, which amounts to correction of the lingual movements. The former uncertainty and hesitancy are supplanted by conscious certainty, based upon knowledge. This psychotherapeutic aspect of the exercises is usually overlooked or underestimated. "Psychic depression disappears under the influence of better achievements."

A. Gutzmann summarizes all the important points in a dozen rules. Liebmann rejects separate exercises and at once starts with the ordinary language, to be spoken slowly, without rhythm and with long vowels. This is quite possible in a number of cases. But that is no reason why exercises should be entirely discarded which, in my opinion, are absolutely necessary in a number of cases. Liebmann's general advice, however, is good, viz.: selection of the material from school books, no scolding, getting accustomed to strangers and new surroundings while speaking.

The *treatment* will occupy at least two to three months, and often

more. After the regular instructions have terminated, the child should be seen from time to time to incite him to progress. Intermittent relapses occur often, but are of no importance.

It is unintelligible how Liebmann can find an average course of four weeks sufficient, and Gutzmann correctly observes that "it is, of course, impossible to correct in two to four weeks a faulty coördination which has existed for a number of years and to establish a new coördination instead." The daily exercises, however, should not exceed one-half to one hour.

As to the results of the treatment, Gutzmann reports cures in about 80 per cent. of his private patients, failures in 2 per cent. and improvement in the rest. The relapses which have come to his knowledge gradually receded from 10 to 2 per cent., ever since he has introduced the system of further control. In his public courses (comprising 1390 cases) there were 72 per cent. of cures, 23.6 per cent. of improved and 3.7 per cent. of negative cases. Troemmer, who has treated twenty adults with hypnotism, has only had 20 per cent. of cures. Ziehen doubts the success of treatment altogether.

Whenever parents are so much wanting in intelligence as to become impatient about the slow progress or apparent initial failure and to interfere with the system of instructions, threatening, scolding or punishing the child, and looking upon every mistake as a relapse, then of course all therapy is powerless and it is better to abandon the case if such resistance cannot be overcome. Few people are aware of the fact that stuttering is not an evil habit, but an affection which it is very difficult to cure. For this reason they prefer the service of quacks who make a verbal promise of a complete cure in the shortest possible time. Instead, however, of entering into a binding contract to that effect, they merely produce a statement for services to be rendered, according to which the victim has to pay an exorbitant sum for a certain number of lessons and a queer-looking apparatus. Whoever is not versed in the law will not notice the trick. There will be great difficulty in proving an intentional fraud and the only remedy at the disposal of the victim is to attempt by judicial proceedings to have the validity of the contract set aside as having been obtained under false pretences and being against equity.

Prophylaxis.—In order to prevent stuttering, it is necessary to superintend the lingual development from the time when, aside from lallation and playful production of sounds, the imitation of our voice commences. Occupation of the child, explaining good picture books, adapting our language to his level, but not to his unskilfulness, are the means to prevent an undue disproportion between a desire to talk and the phonetic and syntactic ability. As the time approaches where a

number of words reach the threshold of language and intelligence, the child will unconsciously make a selection from the bountiful material at his disposal, for which partly phonetic difficulties and to a still greater part the requirements of his stage of development are the deciding points (see p. 366). It is necessary to provide for a large material to select from, because the child can only comprehend certain parts of what is said. "The child must have water to swim in." This material, however, should be within reasonable bounds and adapted to, not imitating, "the phase of development in accentuation, rhythm, gestures, vocabulary, form, and syntax." Therefore, children should be spoken to not after the manner of nurse girls, nor with the mistakes of infantile talk, nor in literary perfection, but slowly, musically, distinctly, in short, simple sentences. Dialect is not objectionable; even the sounds of dialects are pure after their kind. According to Stern, the material should be so confined that "by its multitude it will not disappear from the mental vision and stifle the impulse to imitation."

But what shall we say if a four-year-old child—of course, an only one—whom I was called in to see on account of stuttering, is able at the age of 3½ to recite the first verses of one of Schiller's poems, knows by heart several songs and some particularly difficult words, with which to express himself—at Goethe's time one would have said, to prostitute himself—after dinner! And many sounds could only be stammered. Such silly, parrot-like performances, without the slightest mental foundation, are unfortunately called "charming" in certain strata of our society.

Games with simple little verses or songs, such as are provided by Froebel and others, serve a better purpose and are better liked. Gutzmann advises not only to superintend the development of speech, but also muscular skill, especially the muscles of articulation. It is a pleasure for children to learn clicking sounds, humming with the lips, blowing (on a watch), etc. Great caution is in order where there is stuttering in the family.

After a child has once commenced to repeat sounds or syllables and then stops at difficult words, he should be helped by slow and correct enunciation, thus inciting him to considerate speaking. This can be best accomplished when looking at a picture book or playing. It would be entirely out of place to laugh at their undeveloped skill, while they make every effort to wrestle with the difficulty. By telling little stories and having them repeated, the commencing defect will slowly disappear. In connection therewith, attention should also be paid to correct respiration. All theoretical exercising and school-mastering is wrong. For this reason, Liebmann correctly warns against fathers and teachers who have read books on stuttering.

Stutterers, stammerers, and splutterers in the vicinity are a great danger for predisposed children. True, they cannot always be removed, especially when the child's own parents are concerned, and to remove the child from the family will only rarely succeed. Intercourse with stuttering playmates, however, should be curbed.

The neuropathic basis of stuttering likewise requires careful prophylaxis, in regard to which I must refer to Thiemich, *l. c.* Unfortunately, the physician is often called in too late, after the parents have made one mistake after the other. I will here only brand one of the latter which is particularly pernicious for stuttering children, and that is the fright inculcated by threatening with the "black man" and similar pedagogic monstrosities. (Compare Bendix, this Handbook, vol. i, p. 226, and Pfaundler, vol. ii, p. 233.) For prophylaxis in the school, see Chapter V.

2. DISORDERS OF SPEECH AT PUBERTY.

Stuttering at the time of puberty is chiefly influenced by approaching maturity, and it has been thought well to review the characteristic forms of that period with special reference to prognosis and therapy. Lichtinger writes: "If stuttering has occurred at the time of puberty, it becomes a matter of importance to pay particular attention both to the defect of stuttering and to the genital system." The striking fact that stuttering disappears in girls at the time of puberty, while in boys it not only fails to disappear, but may even originate at that period, is explained by Gutzmann by the fact that girls develop at that time the costal type of respiration which is controlled by muscle and will. Furthermore, the greatest increase in height in young women occurs three years earlier than in boys, in whom, consequently, the manifestations of puberty with considerable laryngeal changes and vocal disturbances coincide with the greatest increase in height at the age of fifteen. (For further details, see Seitz, this Handbook, vol. ii, p. 111.) Injections will occur at the vocal cords of boys which may attain to such high degrees as to assume the flesh-red appearance of acute laryngitis. They cause disturbances of the voice which may give rise to spastic disorders of speech owing to vocal difficulties.

Acute stuttering in puberty of this kind was first described by Gutzmann, who then mentioned seventeen cases.

Symptomatology.—This form of stuttering does not really differ from the ordinary form, but it is characterized by the sudden onset and the prevalence of psychic symptoms. It very often sets in with a short period of aphasia which is caused by a failure of the vocal organs to act. Whispering is also disturbed in a peculiar manner. Stuttering in puberty is been more frequently observed in boys than girls, while the reverse is the case in stuttering due to hysteria.

In one of Gutzmann's patients, a young woman of seventeen, the disturbance suddenly appeared under the picture of aphthongia, after the voice had already been disturbed by puberty. Speech set in with a retching movement of deglutition, during which speaking and whispering were impossible. The spasms ceased after two minutes, when the patient was able to speak fluently, but hastily and with spluttering. At the same time there was disturbance of the voice. Gutzmann's seventeen cases included only one girl. There is, besides, a case of A. Schwarz, a girl of thirteen, which has also been reported under the heading of aphthongia. Another case was reported by Weidemann, of Ebstein's clinic, as stuttering aphasia which occurred in a girl of fourteen, who formerly spoke normally. The onset of aphasia was quite sudden, except in singing, which was not disturbed. There was no dread of talking. Frontal headache and some vomiting. No other findings. Rapid cure.

The *diagnosis* is somewhat difficult in this period, because hysterical conditions have to be taken into account, unless the entire manifestation of stuttering at puberty is to be classed as hysteric. Bruns seems to include all these cases in mono-symptomatic hysteria. In one of Gutzmann's cases the disorder occurred as first symptom of an affection of the cerebral cortex or cerebral tumor.

The *prognosis* is favorable. It would be wrong, however, to rely upon a spontaneous cure, as permanent stuttering may develop after all, unless measures are taken to prevent it.

Treatment.—Treatment is practically the same as in ordinary stuttering, but the psychic aspect is of particular importance. In boys the correct deeper timbre should be practised. Weidemann reports a case which was cured in a week under rest in bed in the hospital(!) and laxatives.

As to *prophylaxis*, the above remarks again hold good, but the vocal disturbances of puberty should be taken into account (see p. 468).

Aside from real stuttering, there is often at the time of puberty a certain laxity of articulation (fatigue) which impairs the distinctiveness of the language. Rapid speaking easily leads to stumbling, which is practically analogous to spluttering. There is also distinct vacillation of the voice. Caution is necessary not to confuse these manifestations with incipient bulbar symptoms.

Lingual abulia in puberty is, according to Liebmann, a disturbance which should be included in expectant neuroses of speech. A girl of thirteen was suddenly deprived of speech, and there was a sensation of angor. Enunciation of the first word was difficult. The first menstruation occurred at the age of fifteen, at which time the same manifestations occurred in writing. She could not write on a sheet of paper placed before her, but was able to do so on a slip lying by the side of it. Later,

she underwent the same experience in piano playing. Otherwise, Liebmann found nothing pathological and, above all, no spasms. The patient was "psychically absolutely normal" (?).

Treatment by practising yields good results in these conditions, which, besides, have also been described in younger children. The differential diagnosis has been dealt with in the following chapter.

3. DISORDERS OF SPEECH IN FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM

These disorders do not present a complete symptom-complex, and their pathology and therapy may therefore be dispensed with. But a description will be given of the disorders of speech in chorea minor, epilepsy, *maladie des tics*, and hysteria, as well as in congenital psychosis (weak-mindedness), following the order of functional diseases of the nervous system as described by Thiemich in vol. iv of this Handbook.

4. DISORDERS OF SPEECH IN CHOREA MINOR

The motor disturbance in chorea is characterized by irregular spastic respiration (atactic respiration), the words being forced out and suddenly interrupted, "cut in two, as it were" (H. Stern). These disorders are more striking when the diaphragm is strongly involved; the respiratory curves are then similar to stuttering.

The voice is weak and fails easily. Retention of the same pitch and intensity is impossible. Twitching of the laryngeal muscles is but rarely observed.

Disturbance of articulation dominates the picture, especially spastic movements of the tongue which cause clicking and sipping sounds. The tongue may be thrown about in the mouth. These involuntary movements lead to disorders of speech that have some similarity with stuttering, but render speech less distinct. It may be severe enough to prevent children from speaking altogether for a time, which would really amount to anarthria on the basis of peripheral arrest of motion.

5. DISORDERS OF SPEECH IN EPILEPSY

Probably the longest known symptom is motor aphasia in the form of aura and of post-epileptic disturbances of uncertain duration. Word-deafness, which in light cases may be confused with dysacusis, occurs less often.

Wildermuth reports a case of complete sensory aphasia in a nine-year-old epileptic. I observed in a boy of the same age partial word-deafness of a changeable extent, which became worse after a paroxysm. There was also literal ataxia owing to disturbed innervation of the *facialis* and *hypoglossus* regions, which improved after practising on

each occasion. The child was regarded as hard of hearing and could understand speech better when simultaneously reading off the face, but nevertheless could understand whispering at a distance of at least thirteen to seventeen feet, provided he was familiar with the subject. There was slight hesitancy in repetition. The latter manifestation caused the mistaken diagnosis of dysacusis in another epileptic boy of six years, who suffered from *petit mal*, and stopped after the first figures repeated at the examination; he then failed to hear any more. Nor did he understand whispered words which were of no interest to him, while words like "Christmas tree" and "apple cake," pronounced in cheerful accents, were correctly repeated at a distance of seventeen feet.

Amnestic aphasia with paraphasia has so far only been described in adults, but echolalia may be supposed to occur in gravely epileptic children, also pronounced perseveration, meaning arrest in the process of perception. Verbigeration with primordial delirium is a "psychopathic disturbance in which patients repeat to themselves meaningless phrases in the apparent character of speech" (Kahlbaum). This does not seem to occur often in children.

There may be disturbance of speech, rhythm and accents of speech which then sounds drawling, heavy, monotonous, or tired. "The epileptic sticks, where the paralytic stumbles." At the same time, there is literal and syllabic ataxia as a sequel to disturbed coördination of the articulating muscles. In children there will be permanent dysarthric disorders following epileptic aphasia. Similar disturbances, however, also occur after bromide poisoning. It is questionable whether genuine stuttering will occur in consequence of epilepsy, although there are stuttering epileptics whose lingual defect is exacerbated after each paroxysm, or it will return if during the intervals it had disappeared.

The monotony of the voice in epileptics has been described by Scripture as an "epileptic voice sign." The vowel curves, which normally rise and fall according to the changes in the dynamic accent, are much more uniform in epileptics. Thus the intensity of the voice does not vacillate in any one particular vowel.

6. DISORDERS OF SPEECH IN *MALADIE DES TICS* (FACIAL SPASMS)

Some details on the relationship between this disease and stuttering have been given on p. 422. Von Sarbo mentions a few more peculiarities as to the manner of talking affected by "tiqueurs." They are in the habit of interpolating stereotyped words or sounds from time to time, which, however, is also observed in other nervous individuals. Small words like "well," "and then," which are used by stutterers to get over a difficulty, do not occur often in children. I was struck, there-

fore, when a stuttering boy with facial tic used the same with great frequency. The real tics, however, are words or sentences exploded under compulsion, which are perfectly meaningless in connection with the sentence they interrupt. Furthermore, these patients are in the habit of repeating words, exclamations, and sentences under compulsion, in spite of the greatest effort to repress them. There is also compulsory enunciation of unarticulated sounds or sound-combinations, or over-extension of certain vowels, and in children, though rarely, there occur compulsory exclamations of offensive and indecent words (copralalia).

7. DISORDERS OF SPEECH IN HYSTERIA

(1) The principal disorder of speech in hysteria is the sudden stop after a shock or paroxysm. A distinction has been made between hysterical aphasia and hysterical mutism. Individuals suffering from hysterical aphasia can usually utter a few syllables, they are not aphonic and exhibit but slight mimic. There are often defective intelligence and objection to writing. Those suffering from hysterical mutism, however, can neither form sounds, nor can they whisper, and are therefore absolutely aphonic; their mimic, intelligence and writing ability, however, are neither arrested nor disturbed.

Saupiquet has shown by a large number of cases that hysterical aphasia may imitate the genuine form in nearly all points (alexia, agraphia, paraphasia), which, however, is relatively rare in childhood. According to him, the expression "mutism" should only be applied to depressive hysterical aphasia (*mutisme dépressive*), the very affection which in childhood seems to occur more frequently than the other forms which only resemble genuine aphasia.

Dumbness occurs quite unexpectedly in boys as well as girls, for instance at night, and disappears just as rapidly or changes into whispering or stuttering. "The patient is more dumb than one suffering from aphasia," says Oppenheim, but he can read without difficulty.

A case of Schubert's (Siemerling's clinic) is remarkable. A boy of thirteen, who had spoken both Danish and German, knew only Danish after recovery from influenza, he having completely forgotten his German. There was also hyperalgesia. Suggestive therapy with faradization and methodic exercises effected a rapid cure.

Gutzmann called attention to true hysterical mutism in typhoid fever.

(2) Hysterical stuttering, or, as H. Stern has it, "stuttering in hysteria," is not distinguished in any way from ordinary, genuine stuttering and has, therefore, to be diagnosticated from the anamnesis and the presence of other hysterical symptoms. It is a rare disturbance and occurs preferably at puberty, so that it will be difficult to differentiate

it from stuttering of puberty. It is caused by "primary disturbance of innervation of cortical motor function or by pathological arrest of the impressions" (Gutzmann).

It also occurs suddenly and often as an early or consecutive symptom of hysterical mutism and, judging by the literature, occurs more frequently in girls than in boys, while in stuttering of puberty the reverse is the case. Spasms can be found in all the regions of the musculature of the organs of speech, just as in ordinary stuttering. Binswanger states that hyperalgesia is never absent, even if no other symptoms of hysteria can be found. Intermissions seem to be rare.

It would seem that authors call stuttering in girls at puberty hysterical, and that in boys stuttering of puberty.

Greidenberg observed hysterical stuttering in a girl of thirteen, who stuttered when the conversation turned upon her defect, but never when indifferent things were the subject of conversation. Von Sarbo observed in one of his cases stuttering while whispering and singing. He also calls cases of aphthongia mono-symptomatic hysteria except where there is ordinary stuttering. Gutzmann reported the case of a boy of thirteen who did not show any spasm during the period of aphasia which followed any question put to him, but there was increased frequency of respiration up to three times the normal, increase of pulse frequency, and perspiration.

The *prognosis* is generally good.

Treatment.—If hysterical stuttering is immediately recognized, the treatment will be promptly successful. (As to its principles, the surprise and neglect methods, see Thiemich, vol. iv, p. 365.) Generally speaking, the former is preferred, with the aid of vibratory massage or faradization. Competence and energy must direct the exercises.

(3) Dysarthric disturbances, syllabic stammering as in paralytics, is very rare; so is snuffling due to paresis or paralysis of the velum. Other forms of stammering do not occur.

(4) Hysterical aphonia is really a disturbance of the voice, but is included at this place because of the interesting relationship. It suddenly occurs in children of seven to fourteen years of age. They will talk in a whispered voice, sometimes they can sing, they cough loudly, when affected or dreaming they speak with a clear voice, but later relapse into whispering.

For treatment, see Thiemich, *l. c.* Mention, however, may be made of Gutzmann's statement that "a favorable result will be attained in every case of hysterical aphonia, even the most obstinate," by systematic physiological practising, similar to the treatment of stuttering, which means transition from aspiration to whispering and then to practising of the voice. Relapses are no rarity.

(5) A peculiar disturbance of speech, resembling bradylalia, occurred in Pfaundler's clinic. An eight-year-old girl suffered from astasia-abasia, aside from hysteria. She had lost her voice in the third week of illness and accustomed herself later to shouting out the words with explosive force in a high key and with lengthened vowel sounds. The disturbance seemed to be voluntary, as was observed when asking for the names of certain objects and when she talked to other children and her parents. Two weeks after admission she answered in a normal voice to an accidental question, after which the disturbance stayed cured. The walking disturbance had already been previously improved by faradization.

(6) Irritation with coughing and interruption of speech, isolated spasms of the muscles of respiration, articulation and the larynx, as well as imitation of animal sounds, are rare symptoms of hysteria.

8. CONGENITAL AND EARLY ACQUIRED DISORDERS OF SPEECH

A large number of imbeciles are endowed with more or less pronounced disturbances of speech of whatever etiology or degree. As a rule, they are independent of any special factors, but rather are anomalies of the organs of articulation, partial or total arrest of lingual development or direct manifestations of psychic disturbances. The act of speaking is less dependent upon their intelligence than lingual understanding. Sollier's idea that idiots are imperfectly and the weak-minded abnormally developed does not apply to speech, either. In describing the disturbances of speech, all attempts at a division into various forms of imbecility may be discarded, except for the thyrogenic form in regard to prognosis and treatment. Further details in this respect have been described by Siegert, vol. iii of this Handbook, p. 551. Besides, mongoloids are capable of a certain amount of education, about which Siegert has written in the same chapter, p. 558. For idiocy and timidity, see Thiemich, vol. iv, p. 382.

The frequency of disturbances of speech in imbeciles and weak-minded may be illustrated by the following figures: In schools for the weak-minded, Doll found 18 per cent., Laquer 24 per cent., Schlesinger 30 per cent., Cassel 33 per cent., Goerke 38 per cent., of the children to be afflicted with defects of speech. Personally, I found thirty-five in fifty-six mentally impaired pupils, or 62.5 per cent. The difference in the figures is probably explained by slight defects, such as light forms of sigmatism, being sometimes overlooked. The lingual development of these children is usually backward and does not keep step with the mental development. According to Cassel, 56 per cent. learn speaking in the second and third years, 44 per cent. after the third year; according to Schlesinger, the figures are 53 and 47 per cent. respectively. Cassel states that children with disorders of speech do not learn speaking until

the third year. Knopf did not find more than 30.5 per cent. of normally speaking idiots in a total of 105. Piper compiled 3931 collected cases and found that 7 per cent. of weak-minded and idiotic children stutter, 25 per cent. stammer and only 13 per cent. lisp (?).

Although the disorders of speech prevailing in the weak-minded have already been described, they may here be reviewed with special reference to the psychic defects in order to obtain a complete picture of these anomalies which are important for pediatricists and school physicians alike. In order to understand the same, the lingual development of normal children, and especially the necessary preliminary conditions, should be present in mind. Partial or total absence of the latter prevents lingual development and is responsible for dumbness of imbeciles up to the fourth or sixth year, or even beyond.

9. MUTISM OF IMBECILES

Acquired dumbness, or aphasia, is differentiated from congenital absence of speech or *mutitas idiotica*. Griesinger says that "these children do not speak, because they have nothing to say." This is no longer correct at the present day. Speech does not only serve the purpose of communication and designation, but also that of volition, and the latter, the perception of requirements, is hardly ever absent even in idiots. Idiots must be on the lowest possible level if they do not even express hunger, although there may be lallation and crying for a whole night both before and after feeding, without any participation of the sensory sphere. The rest of the imbeciles are able to express desires by the timbre of lallation and gestures, especially those expressing displeasure, even though the development of their gestures is very slight and late. So there must be other reasons for their failure to speak. These consist in the absence of the preliminary conditions, less of the sensory spheres, but rather of motor incitation. What is missing is the primitive motor impulse which, in the stage of lallation, is the decisive factor for playing with the organs of speech. There is no impulse to imitation, nor its premises: concentration and memory for sensory impressions and movements. Their psychic life, so far as such exists, is surcharged with impressions of dislike, and their desires are not always strong enough to overcome the apathy and resort to lingual utterances. It is for this reason that the motor performances of these children are defective in the preliminary stages of speaking, lallation and the first imitations, and consequently their future understanding of speech will be slight or nil.

Frequency.—Mutes are not particularly rare among idiots; according to Knopf, there are 7 per cent. of boys and 11.3 per cent. of girls. Wilde has collected 5180 mute cases, 433 of whom were not dumb. 242 of these were idiotic.

Variety of Form.—A separation of mutism into various groups is justified according to the nature of the disturbance, because the dumbness is usually not complete and its prognosis depends upon various factors, notably the behavior of the perceptive sphere.

(1) Thoroughly apathetic children sometimes have a primitive understanding of speech, supported by gestures, but they will not utter sounds except some whispered, indistinct lallation.

In one of Frenzel's cases, speaking could not be induced, unless the teacher accompanied the repetition, or upon tactile incitation by touching the arm or mouth. Winckler reported the case of a boy who could not be made to speak except on seeing the optical picture on the black-board.

(2) Psychically deaf imbeciles make themselves understood by signs. Speech, sometimes even its modulation, is incomprehensible to them, which may be the consequence of failure to perceive the words (Heller) or of defective attention and memory (Gutzmann). They are impressed by musical sounds, however.

There are often remnants of stammering, meaningless echolalia, as for instance in the six-year-old boy, reported by Liebmann, who could not distinguish entirely different noises owing to complete absence of concentration. Aside from this psychic deafness, there are a number of characteristic symptoms showing a low mental level.

These cases are distinguished from organic sensory aphasia and auditory dumbness in normal or almost normal children (p. 388) more by their general behavior than by isolated symptoms. Careful observation is required to prevent confusion with deaf-mutism.

(3) The versatile forms are the opposite to the apathetic. These children are always gay and romping about, but they react but slightly to acoustic or other external irritations. The reason of the latter manifestation is not a primary disturbance of attention (maniac nature), but that they are incapable of receiving any deeper impression.

Sometimes they will learn short sentences, which they repeat to themselves innumerable times in a stereotyped and spluttering fashion, as if impelled to do so. They will even repeat the same phrase in answer to questions, without having the slightest idea of its meaning. They may understand a few words and commands, but only as the result of mechanical training, not of logical comprehension (see p. 390). These cases are not rare in grave mongoloid idiocy.

(4) In a further group the real intellectual disturbance is less grave, but there is no intellectual interpretation. These children understand speech, more or less, but they can speak only a very few, if any, incomplete words and those without correct intonation. It is usually impossible to make anything out of them at the first examination, and their "vocab-

ulary" will only reveal itself in the course of time. They will remain stationary on Meumann's volitional or associative-reproductive stage. Liebmann believes in a stupendous clumsiness of the organs of speech. This is partly correct, as enormous muscular efforts can be observed for the production of imperfect words. It also often happens that pathological changes in the peripheral organs of speech aggravate speaking itself, such as anomalies of the maxillary formation, a thick, heavy tongue with slight mobility and enormous hyperplasia of the lymphatic pharyngeal ring. In others, however, there seem to be pronounced arrest of psychic development and absence of rapid associations, which are shown by relative inactivity and an unusually low voice. The latter is less attributable to timidity than to inactivity of the mind.

(5) Weygandt and Heller have described *dementia infantilis*, which sets in during the third or fourth year and leads to idiocy. Spontaneous, and later all, talking becomes continually poorer and less coherent, until finally it is completely lost.

The disturbance may commence with stuttering, followed by changes of sounds, such as *mimmi* for *mamma*. Objects pointed out to them are designated by unintelligible words, probably mutilations of formerly correct expressions. Words in well-known children's poems may at first still be supplemented; the enunciation of words in singing becomes less distinct; they are "peculiarly stretched and maltreated," until at last only a few words remain, such as *papa*, *mamma*, *water*. Verbigeration of meaningless words also seems to occur. Heller states that this disorder of speech may be improved by exercises.

The division into four groups is of course only valuable as a symptomatic outline which has been made without reference to the etiology. Consequently, there are many cases which do not at once fit into any of these groups. The lines of demarcation are blurred and it is only the dominant signs which permit of such a separation. Genuine aphasia is described on p. 460.

Diagnosis.—It is necessary to analyze all the lingual and mental faculties in each individual case, aside from a general physical examination, in order to find out in what point the patient chiefly deviates from the normal.

The so-called test method which was first used in 1883 by Galton, in England, to examine into the mental faculties of adults, has yielded the best results in this respect. It has since been amplified and improved by numerous authors. A detailed criticism of the same has been published by Meumann in an article on the test of intelligence in children attending public schools, contained in vol. i of "*Experimentelle Pädagogik*, 1905. Albert Liebmann, in 1898, compiled a routine method for the examination of mentally deficient children. The examination is

conducted with employment of the simplest possible means, in order to find out the children's capabilities in the acoustic, optic, tactile and motor spheres. The first point is whether there is reaction to acoustic irritations at all, and whether sounds and noises can be distinguished and possibly localized; whether single words and sentences will be perceived, simple commands executed, whether forms and colors can be recognized, sizes differentiated and whether there is orientation as to objects in space. Many children can recognize by their tactile sense what they cannot distinguish by the ear. Differences in weight and temperature are not as readily perceived. Simple movements and action should then be tested, as for instance walking in various ways, movements of the arms and fingers. But there is no use denying the fact that even these tests may amount to complicated efforts on the part of the child. In regard to perception of noises, for instance, we do not only test the hearing acuity, but also the attention paid to acoustic irritation. If all kinds of sounds are produced before a child with bandaged eyes, or behind his back, as for instance rattling with a bunch of keys, striking a gong, manipulating a pair of scissors, it is not only the hearing acuity and attention that are under test, but also the power of retention. It is perfectly possible that from the moment of acoustic perception to that of optical perception, when the objects are seen, the acoustic impression has long been forgotten. Furthermore, a sufficiently retentive memory is presupposed to enable the child to remember from previous experience what kind of noise is produced by a certain article. If it is desired to facilitate this memory test, or if a sufficiently good memory is not expected to exist, it will be necessary to acquaint the child with the noises to be tested. At the same time an idea may be gained in how far the interest of the patient can be aroused. All these possibilities have to be kept in view. As these children are usually below school age, they should not be expected to know things which might even puzzle a normal child. The results of these tests should be compared and controlled by frequent examinations and observations, as these may vary on different occasions. No doubt, this is time-absorbing, but it is the way by which it may be possible to get an insight into the psychic capabilities and defects and to find out in what sphere a child is most capable. As to the language itself, the extent of its understanding should be determined, as far as may be possible, likewise the ability to repeat and designate. This will also reveal disorders of speech as well as central disturbances, as for instance perseveration.

The *prognosis* depends upon the result of the entire somatic and psychic examination. Given appropriate treatment, speech will develop, at least to some extent, in most cases, but it will require years to achieve what normal children can accomplish in a few months. The prognosis

of the fourth group is better than that of the first three; among the latter, the first two have better chances than the third, because it is easier here to promote retarded development than to create arrests and compensate to some extent the defects of attention. The mongoloids again have better chances than the other versatile forms, because they can be trained within moderate limits, so that they may at least be able to speak intelligibly, even though their grammar and syntax will not be perfect.

The *treatment* of the various kinds of mutism in imbeciles depends upon the kind of disturbance. It can only be pedagogic, must commence very early and adapt itself to the normal development of speech. The object is to enable children to make themselves understood and to arrive at a certain mental development by which they can make themselves useful in a modest way.

Generally speaking, children should be incited to repeat names of objects, taken from their (small) sphere of interest, which have been repeatedly enunciated before them with a loud, clear voice. Questions should be immediately answered by the examiner himself, so as to incite to primitive repetition. It is of little use extending a lesson over a long time, but its constant repetition on every possible occasion is useful. S. Heller advised to let the children lie flat on the floor, so as to have them in a quiet position. Apathetic children, however, should be incited to movements. Although the first phonetic elements presented to imbeciles should consist of words, even though they be not understood, it should not be forgotten that in the period of lallation the playful practice of articulation was insufficient. For this reason it is necessary to combine the exercises in articulation with gymnastics to the greatest possible extent. The latter include simple exercises of the muscles of the maxillæ, mouth and tongue, such as opening, closing, pointing the lips, blowing, tongue movements, etc., also respiratory exercises such as were described for stutterers. This is followed by combination of the physical movements with respiratory and sound-exercises, nor should attempts at writing and reading be forgotten. The object of these exercises is the development of motor skill, attention and will power. "The physical exercises involve not only physical, but also nerve gymnastics, perhaps even more so" in the sense of training the entire nervous system (Du Bois-Reymond). Spitzzy recommends gymnastics with apparatus for the physical training of the imbecile. The mental lessons are given at first with the aid of palpable objects, clay figures, etc., which will later be replaced by picture books. Heller advises to awaken the interest of psychically deaf children by the sounds of musical instruments, which they will learn to differentiate and locate. This is followed by repeated enunciation of various objects, at first

answered, as for instance "What is your name?"—"What is your name? Yes, Otto." Similarly, parts of the repeated question may precede the answer (partial echolalia), as pointed out by Maupaté. The latter forms rather correspond to thinking aloud. Words repeated in echolalia usually have the same intonation as our own language, but may also deviate and become monotonous, failing to embrace the lingual accents.

Among the idiots examined by Knopf, 2.3 per cent. of the girls and 6.5 per cent. of the boys spoke in echolalia.

12. STAMMERING OF IMBECILES

This typical defect of speech in the imbecile is usually the result of retarded lingual development and may also occur as written stammering in older children. Knopf found stammering in 64.5 per cent. of the girls and 29 per cent. of the boys at Idstein, Westergaard, in 42.4 per cent. among 250 demented.

The causes are generally the same as in functional and mechanical dyslalia of normal children, except that there are organic defects of articulation, as for instance dental anomalies owing to deformation of the maxillæ, to a considerably greater extent. According to Herfort they amount to 23 per cent., in my own fifty-six cases to 62.5 per cent., all forms being represented in about the same frequency: open dentition seven, dental arches seven, cross dentition seven, prognathism eight, progenia six. These deformities cause enormous difficulties which a normal child might wholly or partly overcome. Further characteristic defects are the following: Functional open snuffling as an expression of weak innervation, and laryngeal dyslalia, as for instance the rough and deep voice observed in mongoloids.

Another characteristic sign is the prevalence of syllabic and word stammering, and Hottentotism, which proves that the lingual defect is not to be regarded as a complication of dementia, but as an arrest of lingual development owing to central defects, chiefly of the motor sphere and concentration. A few sounds are usually formed correctly or nearly so, but when combined into words, everything is blurred, chiefly owing to numerous elusions and consequent mutilation of all words which are cut down to one or two syllables. This condition is still grouped as stammering for want of a better term, although it should not be overlooked that these defects of speech again arrest the psychic development, inasmuch as they limit the acquisition and use of the language to a minimum, depriving the child "of the most important tool to perfect the process of thought" (Wundt). These cases, therefore, present a very unfavorable aspect, and only the success of correct treatment will show that a more favorable prognosis would have been justified.

The *treatment* is the same as in stammering of mentally sound

with a very loud voice. Imhofer also advises the employment of music (singing), based on his examinations of the musical hearing ability of mentally impaired children. It will be well to remember this suggestion in the treatment of mongoloids with a memory and interest for music.

General dieting should be in the foreground of the treatment, according to Gutzmann. It would be wrong to give way to the whims of these patients in regard to quantity and quality of the food or to the manner of eating and drinking, which is usually one of the parents' mistakes. The more they get accustomed to order, cleanliness and obedience, the better will their progress be in lingual education. Where there is a disturbance of the thyroid function, the administration of thyroïdin is indicated (see Siegert, *l. c.*). In many apathetic imbeciles with different etiology there are also occasionally slight manifestations of dysthyroidism. If they are fed carefully with thyroid preparations, they often commence talking at an early date. Generally speaking, the only difference between apathetic and erethistic imbeciles is that the "eruption of the speaking ability" occurs rather suddenly in the former, and only gradually in the latter. The faucial tonsil should never be removed for a mere disorder of speech, unless it also causes other manifestations. This operation will not improve speech, while the psychic trauma may have a very injurious effect.

10. DISORDERS OF SPEECH IN PARTIALLY DEAF IMBECILES

If dysacusis occurs in conjunction with central defects, weak minded children will, of course, learn to speak late, there will be considerable stammering for a long period and the tone is generally very low. Retardation in lingual development alone would not justify special description but for the valuation of the two components, peripherally impressive and the central, which are of diagnostic prognostic interest.

Doubtless there are many hard hearing patients in institution idiots and schools for the mentally unsound. Brühl and Nawroth found 28.5 per cent., Nadoleczny 58 per cent. and Wanner 69.1 per cent. of cases with about 23 feet hearing distance for whispered voice. Some of these children may have been sent to the special schools on account of their impaired hearing, but others are undoubtedly mentally imbecile. In the latter, treatment of the ear alone would have no material influence upon the lingual and mental progress, not even in mongoloids whose palatal formation often simulates the adenoid type.

It is evident that in later years disorders of speech are particularly frequent among the demented whose hearing ability is impaired. The opinion as to whether these disorders are linked with the primary affection can only be formed after prolonged observation.

children (see p. 397), but it requires more time. It may, however, be mentioned that the removal of the faucial tonsil will not influence the psychic condition, even if other conditions should justify its removal.

13. AKATAPHASIA OF THE IMBECILE

The defect of intelligence of not being able to construct a sentence according to grammatical and syntactic rules permanently dominates the picture of a great number of imbeciles and may therefore be designated as a typical dyslogical disturbance of speech. Knopf found this condition in 16.3 per cent. of girls and 25.8 per cent. of boys. All forms of akataphasia will occur (see p. 394). The relative frequency of verbal akataphasia is noteworthy so far as it results from the inability of rapidly designating an object or rapidly associating the object with its name. This leads to mistakes like "nickel" for "apple," the coin being mentioned with which to buy the article. The desire of possession plays a part; or, a "carriage" may be circumscribed by "what to ride in." Idiots will rarely acquire fluent speech, even if the construction of sentences does not remain stationary at the level of a three-year-old child. The slight range of the initial vocabulary is the expression of the narrow sphere of interest, in which practically nothing else finds a place but the desire of eating and playing. Thus these children will for a long time remain in the lingual stage of substance and avail themselves of the one-word sentence or the paratactic sentence of several words. Verbs are acquired next and dominate the so-called infinitive language in the stage of action which, at a relatively late period, is followed by the relational stage with a scanty number of adjectives. Pronouns are understood, but used very rarely, possibly in a possessive and demonstrative nature, which accounts for the character of akataphasia pervading speech in advanced periods, the more so as flexions are learned only with difficulty. Defects of grammatical development, omission or faulty use of nearly all abstracts due to their not being sufficiently understood, are still found in demented individuals who have made a relatively good lingual progress.

Spluttering of imbeciles is not distinguished from ordinary spluttering (see p. 392), but occurs oftener than in normal children. Knopf states that among the idiots he examined spluttering occurred in 11.6 per cent. of girls and 8 per cent. of boys. The disturbances of speech rarely exist alone, they being usually associated with stammering and akataphasia. Genuine syllabic stumbling, as in paralysis, is rare.

14. STUTTERING OF THE IMBECILE

The stuttering of the imbecile differs from that of normal children in that it seems to be more frequently attributable to an organic disturbance of coördination than had previously been assumed. This

would also explain the relatively high frequency, Piper giving 7 per cent., Knopf 9.3 per cent. in girls and 17.7 per cent. in boys. (Further details on stuttering as a focal symptom will be found on p. 456.) The prognosis of stuttering in these cases is usually unfavorable. Stuttering imbeciles may become dangerous if, in their rage for being ridiculed, they commit deeds of violence. This was exemplified in a case before the Children's Criminal Court in Munich. A boy, becoming enraged from being scoffed at, drove a stick into the offender's eye. Death from meningitis followed. The murderer was a demented imbecile with defective hearing who showed neither repentance nor consciousness of the gravity of his deed.

15. DISTURBANCE OF LINGUAL ACCENTS IN THE IMBECILE

The blurred character of the language due to insufficient articulation has already been pointed out under the chapter on Stammering. This is increased in imbeciles by absence of the dynamic accent (p. 377), showing a certain amount of albulia. Monotony and faulty intonation have likewise to be attributed to disturbances of the mind. Knopf observed the remarkable fact that 7 per cent. of the girls and 11.3 per cent. of the boys spoke in an interrupted, hesitating and usually low voice—bradylalia—which is probably explained on the assumption of weakened innervation. Motor anomalies are also often met with. Owing to over-extension of the temporal accent, the syllables and especially the vowels are enunciated very slowly and frequently separated by intervals, with the result of a staccato voice. This differs from the speaking disorders in multiple sclerosis by the fact that a pause does not occur after every syllable or word. Thus, speech consists of a series of interruptions. This hesitation may be confined to certain vowels, so that other parts of speech are continued uninterruptedly (described by Maupaté as *annonnement*). Conversely, the talk of imbeciles is often accelerated by the shortening of the temporal accent, which would unfavorably influence the other two accents and thereby the distinctness of expression. A combination between repeated rapid explosion of words and the stereotyped way of speaking has been described by Maupaté as "*Parole Explosive*," who explains the same as an unbridled discharge of violent lingual excitations. Small word-groups, often contracted, follow each other, separated by short intervals. This peculiar disturbance gives the impression of overcoming the impediment by a sudden impulsive effort.

The *treatment* of all these disturbances is the same as that for stammering and spluttering, combined with exercising the motor sphere (gymnastics). Any defects of the organs of special sense (eye, ear) should be carefully watched.

Disorders of speech in imbeciles, which must be regarded as symptoms of organic affections of the nervous system, will be dealt with in the next chapter.

16. DISORDERS OF SPEECH OWING TO ORGANIC AFFECTIONS OF THE NERVOUS SYSTEM

Disturbances of speech occurring in these conditions are chiefly of diagnostic importance, and they will be discussed in the same order which Zappert has followed in describing the causative affections in vol. iv of this Handbook. As to the endogenous affections of the central nervous system, there come in for consideration spastic family affections, hereditary ataxia and bulbar affections (comp. vol. iv, 157, 160, 172).

(1) Disorders of speech occur as an essential symptom of the cerebrospinal symptom complex of spastic family paralysis, which usually assumes the form of bradylalia. Massalongo speaks of "stuttering" and disturbed tongue movements as first symptoms in three children of the same family, when seven years old, and who later lost speech entirely. Probably, therefore, they suffered originally from dysarthria which finally developed into anarthria. Oppenheim states particularly that spastic conditions will spread to the articulating musculature and that of deglutition, thereby adding "a purely spastic form of bulbar paralysis."

(2) Oppenheim states in his text-book, when dealing with disorders of speech in hereditary ataxia, that the latter did not appear before the late stages, while Friedreich himself states that they occur rather early in the course of the disease. They began "at first as lallations which increase with rapid talking and become worse in the course of years to such an extent as to border on unintelligibility. Even when the speaking disorder had reached the climax, the voluntary movements of the tongue in all directions were perfectly free not only inside the oral cavity, but the tongue could also be protruded and held quiet in any position." He explains the affection as a disturbance of coördination of the articulating movements of the tongue and demonstrated neuritis chronica descendens in the hypoglossus trunks. The words are pronounced "slowly, sometimes with impulsive interruptions." Freud calls special attention to the retarded, slight staccato style. These are evidently forms of dysarthria (literal and syllabic ataxia) which injure the lingual accent as well as the articulation of polysyllabic words in particular. P. Marie observed in his *héréd-ataxie cérébelleuse* disorders of speech at the time the ataxia spread to the upper extremities (which means during the first three years) which resemble those in multiple sclerosis, except that there is a slighter staccato expression. The

muscular movements of articulation are considerably exaggerated, giving the impression of clumsy and troublesome speaking. In other cases a few words can be spoken correctly while sentences are rendered with a certain degree of monotony. The language is sometimes badly articulated and can only be understood with difficulty, but there is neither staccato nor explosion, nor any demonstrable dysarthric or purely motor disturbance.

While experimental, phonetic studies for the last two affections are still wanting, exact investigations have been made into disorders of speech in *progressive bulbar paralysis*, possibly because they are more readily observed and also because they command greater attention as an initial symptom. Prolonged speaking causes fatigue in the first place, later speech becomes more and more indistinct, but may improve for the moment by dint of energetic effort. There occur dysarthric disturbances which were first observed by Goldscheider by means of graphic pictures of the expiring air as it escaped from the nose or mouth.

His curves showed weakness of the closure in all explosive sounds and slow instead of rapid opening which caused the extension of these sounds; they also showed the escape of the respired air through the nose in nearly every sound, explaining the snuffling timbre of the vowels. (Comp. *rhinolalia aperta*, p. 406.) Among the friction sounds, R suffers most, owing to the tongue movements being irregular and weak. The R-curve, therefore, is flat with slight irregular waves, as against the very distinct and regular normal R-curve. The nasal curve, which should really be absent here, is very steep. Boumann has registered the movements of the maxillæ, upper lips, and oral fundus with Zwaardemaker's apparatus. He found vibratory movements in nearly all curves or at least simultaneously in two of them, indicating light consonantal stuttering, as for instance g-go, f-fable, also the nasal curves and open snuffling.

It is a noteworthy fact that distinct sounds, as that of the closure of the velum, are demonstrable by methodical examination long before they strike the listener's attention. The qualitative and quantitative progress of the affection may be observed in the same manner.

The distinctness of speech varies according to the nerves that are impaired. If the lips are involved, a sensation of rigidity will be experienced there, later they become thin, so that they cannot be pointed, whistling becomes difficult and finally impossible. The sounds B, P, M, F, O, and U become indistinct, B, for instance, being pronounced like Mb, P like Mp or V. If the tongue is more strongly involved, the enunciation of D, T, S, Sh, L, R, EE, will suffer, before any particular atrophy or disturbance of the tongue can be demonstrated. Dysarthria

becomes most striking by paralysis of the velum, because the nasal participation of all sounds changes the character of speech much more than does the indistinctness of the consonants. This may go so far as to the labial sounds being omitted entirely owing to paralysis of the velum and weakness of the lips. The voice becomes weak in time and badly modulated. The musical scale can no longer be produced with certainty, singing becomes impossible. The sound may vacillate, especially in the transition from sonorous consonants to vowels and *vice versa*. Finally, the voice will sound like breathing owing to defective closure of the glottis. Toward the end of the affection, dyspnoea will likewise contribute to the difficulty of speaking.

If treatment can be spoken of at all in this complaint, it may be stated that Gutzmann and Knopf have observed transitory improvement of speech and deglutition by systematic exercises (raising of the velum).

In ganglionic aplasia (vol. iv, p. 159), as well as congenital paralysis, the disorders of speech are less pronounced and not progressive. As a rule, none but the labial sounds are spoken badly, and neither whistling nor blowing will quite succeed. Movements of the tongue, however, can usually be executed or partly so; paresis of the velum has been mentioned only once by Hoppe-Seyler. Talking is learned with difficulty.

17. DISORDERS OF SPEECH OWING TO HEREDITARY SYPHILIS OF THE CENTRAL NERVOUS SYSTEM

These disorders have already been discussed, so far as infantile cerebral paresis or multiple and diffuse sclerosis of the brain of syphilitic origin are concerned. Aphasia in meningitis gummosa is discussed on p. 462. For further details see Zappert, vol. iv, p. 184.

(1) Locomotor ataxia and ataxia of the upper extremities are rare in infantile tabes. For this reason, spreading of the latter to the muscles of the face and tongue, which, according to Oppenheim, leads to peculiar disturbances of speech caused by excessive movements of the muscles of the lips, tongue, and maxillæ, is not described in children. In tabetic paralysis, however, literal ataxia occurs. It is a noteworthy fact that the infantile voice will persist beyond the years of puberty owing to the absence of that development.

(2) The disorders of speech in progressive paralysis of children are the same as in adults. These forms of disorder (literal ataxia) are distinct from the bulbar form by the fact that the single sounds are pronounced correctly, even the phonetic examination shows no deviation but the combination of sounds in syllables and words is a failure. ~~Her~~ the designation of "syllabic stuttering." The disturbance ~~increases~~ when a difficult word has to be repeated several times, which is not

case in bulbar paralysis. Boumann has observed in the course of his phonetic examinations slow, irregular vibrations of the curve preceding speaking which reproduced the movements of the upper lips, maxillæ and oral fundus. Quivering of the lips and twitching of the facial musculature are also directly visible. The voice loses its modulation and strength owing to defective innervation. Hesitation due to difficulty of diction and searching for words are characteristic signs and can also be demonstrated in the curves. The disorders of speech are progressive according to the character of the underlying cause: speech is impoverished and becomes progressively more indistinct.

(3) Aphasia and bulbar disorders of speech also occur in connection with diffuse cerebral syphilis.

Gutzmann noticed in two cases of hereditary syphilis that the well-developed language was rapidly lost in the third year of life.

(4) In the case of a brother and sister, speech did not develop before the fifth year for the same reason; it was snuffling and unintelligible, although the velum was not paralyzed. A boy, twelve years old, was intellectually and morally demented; his first teeth erupted when twenty-eight months old; he spoke his first words at the age of four and learned walking at five (severe rhachitis). There were hiccoughing and moderate snuffling owing to insufficiency of the velum. He speaks a few easy words. His spontaneous language is raw, low, blurred due to blending of the consonants and shortening of the vowels, over-haste and unintelligibility. It is therefore a mixture of stammering and spluttering.

(5) I have seen stuttering come and go in another case which was clearly due to organic causes. The rarity of the occurrence justifies a short description of the case. A six-year-old boy had repeatedly suffered from keratitis parenchymatosa, commenced to stutter at the age of five, principally repeating words. In the last three weeks stuttering increased considerably and was of a pronounced chronic character. Simultaneously there occurred numerous signs of cerebral syphilis (which were attributed by Professor Hecker to gummous infiltrates of the cerebral and spinal meninges without any real tumefaction). There were headache, ataxia (which could not be explained by the simultaneous labyrinthal syphilis), fatigue, irritable debility, hyperæsthesia of the lower extremities, especially of the left, retarded conduction in the same region and vasomotor disturbances (cyanosis and cold), reduced patellar reflexes, left more than right; the right ear had become deaf, the left labyrinth partially so; rotatory nystagmus to the left. At last an ulcerating gumma of the left tonsil was discovered. Anti-syphilitic treatment improved the general condition, the hearing distance of the left ear was slightly increased, and stuttering disappeared almost completely in the course of a month. A month later he returned with a relapse of the central

manifestations, which, however, disappeared again under the same medication. One year later, the child spoke very well. Slight imbecility (absence of inhibition) was one of the symptoms.

18. DISORDERS OF SPEECH DUE TO ENCEPHALITIS

The disorders of speech of acute inflammatory bulbar paralysis which have been described above also occur in polio-encephalitis acute inferior (Zappert, vol. iv, pp. 198 and 202), except that their onset is more sudden, and speech becomes more rapidly indistinct. Nevertheless, a complete cure is possible.

Disturbances of speech which occur as symptoms of multiple encephalitic foci of varying etiology are more interesting and more important in differential diagnosis. Their nature is as variegated as the entire complaint. In one case, there may be bulbar symptoms, as for instance progressive bulbar snuffling which may give way for a moment to improved, though vacillating, speech enforced by supreme effort, but then return with increased emphasis owing to fatigue; the number of respirations interrupting speech render the latter quite unintelligible, terminating in a low, lisping squeal. There is distinct vibration of intention of the vocal lips. In other cases the manifestations somewhat resemble multiple sclerosis. Aside from the speech disturbances, there are bulbar, cerebral, and spinal symptoms which complicate the picture. The lips, which at first are merely paretic, together with the tongue and palate, finally become paralyzed, the velum presenting the most characteristic disturbance. I have also observed disturbed respiration in the shape of interrupted explosive breathing. The curves are steep and separated by pauses of one to one and one-half seconds' duration.

19. DISORDERS OF SPEECH DUE TO SCLEROSIS OF THE CENTRAL NERVOUS SYSTEM

The disorders of speech in multiple sclerosis (comp. Zappert, vol. iv, p. 246) in childhood do not occur less often, but later than in diffuse cerebral sclerosis, where speech is often lost rapidly. Slow monotonous speech, devoid of musical accent and often interrupted in the middle of the word by exuberant inspiration, is a characteristic symptom, if present. As a rule, the manifestations occur late and gradually increase. They are seldom complicated by grave bulbar disturbances of phonation and articulation (*rhinolalia aperta*).

Investigations instituted by Goldscheider and Boumann have led to the following results: Retardation of the respiratory movements and articulation, the latter becoming easily fatigued; this is shown by the curve of expiration by elongation and aplanation. The curves of the movements of the lips, maxillæ, and oral fundus show distinct vibra-

tion. The various sounds, the complicated friction and palatal sounds, become indistinct. In this way speech assumes the character of undefined bradylalia, becomes laborious, vacillating in pitch, with a staccato effect which is chiefly dependent upon the respiratory disturbance. H. Stern has graphically demonstrated the interrupted respiration by means of pneumographic curves. The exuberant character of the inspirations is explained by defective opening of the glottis, since the vocal cords participate in the intention tremor of multiple sclerosis. Owing to the rarity of these cases in childhood there is only one observation available (Krzywicki): namely, a seventeen-year-old boy whose vocal disturbance began when thirteen years old. Owing to the effort involved he had soon given up speaking altogether. His inability was striking even in single words. The voice was tremulous and vacillated between thoracic and falsetto voice. The intention tremor of the vocal cords and their pendulous twitching after cessation of phonation could be observed laryngoscopically. Gutzmann called attention to the similarity between this disturbance and stuttering, there being pauses between single words which were enunciated explosively, while longer words are hacked into pieces by intervening respiration. The diagnosis could not be made in some cases except on the ground of other symptoms, etiology and duration of the affection.

The older literature contains two cases of aphasia in girls three and one-half years old with the diagnosis of multiple sclerosis (Humphrey, Pollak). The latter author observed staccato crying in his case.

20. DISORDERS OF SPEECH DUE TO INFANTILE CEREBRAL PARALYSIS

This symptom-complex as the starting-point of various affections has been described by Zappert, vol. iv, p. 252. Aphasia will be discussed in a special chapter, while the other disturbances of the language in cerebral hemiplegia and diplegia, with special reference to general rigidity, bilateral athetosis, and infantile pseudobulbar paralysis, will be discussed at this place. Their frequency will be seen from a statement by Ganghofner, according to which twenty-six out of forty-four cases above two years of age, or 59 per cent., suffered from disorders of speech, in nine cases there were complications by athetotic and choreic movements, and in eight aphasia. A large portion of these children are demented.

(1) As to hemiplegic forms there are, aside from aphasia, which also occurs in hemi-paresis and hemi-athetosis of the left half of the body, remnants of the same, stammering, functional snuffling, and (aphactic) stuttering.

The following case will serve as an illustration. A weak-minded boy of eight learned walking at the age of one, talking at four years, and

had left-sided hemiplegia. While single sounds are formed correctly, there is distinct syllabic and word stammering (as well as paragammacism, omission of prefixes, etc.) associated with grammatic akataphasia of the second degree. His speech consisted of paratactic several-word sentences uttered in an inordinately loud and spluttering voice, interrupted by rapid breathing, with a slight musical accent, sometimes stammering over difficult sound-combinations, provided he takes the trouble of starting them. Thus, for instance, he would hastily repeat: "The book-the book ha ma-ny le-letters."

Similar cases of stammering, combined with snuffling and stuttering in infantile cerebral paralysis, have been published by Oltuscewski, who believes in good results from practice therapy so far as speech is concerned.

(2) In the various forms of cerebral diplegia, similar and partly serious disturbances of speech occur still more frequently. In general rigidity (Little's disease) especially, there is violent stuttering which Kobrak rightly regards as a focal symptom. The constancy of the disturbance, which in ordinary stuttering is but rarely so pronounced, seems to me characteristic. Secondary psychic symptoms (linguophobia, etc.) were absent in the cases I have observed.

A boy suffering from general rigidity, who learned talking at the age of three, and did not commence walking after he had been operated on by Professor Lange in his fourteenth year, stuttered equally hard in spontaneous talking, repeating, reading, reciting, and whispering. Singing, too, was disturbed by the exaggerated dynamic accent. Speech is interrupted by numerous small respirations, several words being exploded at a time; then follow spasms which are overcome by sharply articulated staccato utterance of separate syllables. Sometimes he speaks at inspiration. The entire mimic musculature participates in the act. This is no doubt a case in which, aside from spastic disturbances, there are difficulties in the way of phonation, a kind of syllabic stumbling. Pneumographic examination shows interrupted inspiration and extirpation at rest, similar to multiple sclerosis, and distinct tonic and clonic spasms, notably of the diaphragm, while speaking. Spasms are also said to occur, during which the tongue protrudes far out of the mouth.

There are, besides, the less characteristic forms of stammering, chiefly syllabic and word-stammering with elisions, metathesis, and assimilations (comp. p. 372) in these forms as well as in those previously mentioned and those that follow.

(3) If cerebral diplegia is complicated by bilateral athetosis or chorea, speech is usually interfered with by the severity of the disturbance, particularly so if twitching of the tongue, velum, and platysma occurs. Rigidity and athetotic movements may also involve the maxillary and labial muscles, as happened in a patient of Oppenheim's who

suffered from nearly absolute anarthria. A girl, ten years of age, had great difficulty in talking and had most pronounced dysarthria. A characteristic sign of these pathological forms is spastic bradylalia, laborious slow speech, accompanied by considerable participation of the facial musculature. There are also bulbar symptoms, as for instance in cases of Cassirer and Toby Cohns, who also found disturbances of the voice, consisting in deep voice and aphonia. A case described by Liebmann as a choreic disorder of speech also belongs to this category.

I observed grave spastic bradylalia in a six-year-old girl (an eight-month child), whose voice was low, monotonous and accompanied by peculiar impulsive movements of the head and upper part of the body. Stammering was chiefly confined to syllables and words; sound-stammering occurred only with L and R, which were omitted. The monographic curves showed all manner of spastic disorders of respiration, such as were described in stuttering, while respiration at rest resembled the above case of general rigidity.

(4) The speech disorders of infantile pseudobulbar paralysis deserve special consideration. The paralysis first involves the buccal musculature, the tongue, and in about one-half the cases also the velum, which becomes completely paralyzed. Consequently the labial sounds are either completely absent or indistinct, and the same refers to the lingual sounds P, B, F, V, O, and U, as well as to T, D, K, G, R, and S. This is complicated by open snuffling, which Peritz believes to be due to central causes, if no paralysis of the voluntary palatal movements is demonstrable. Speech is certainly more severely affected than the voluntary movements, and there is no parallel between dysarthria and palatal paralysis. Speech may be confined to rapidly exploded, loud sounds, of which none but "ah" and "ha" can be made out, or it becomes drawling and indistinct, or of a low key if the laryngeal muscles participate. H is then absent, and a difficult sequence of sounds is separated by interpolating vowels, as for instance "perofessor." Laborious, sometimes hasty and explosive speech is interrupted by coördinate spastic movements of the muscles of the maxilla and the tongue or by athetoid movements of the face. The form of stuttering thereby occasioned has been called dysarthric by Abadie, and occurs oftener in the spastic than in the paralytic form of pseudobulbar paralysis.

Since the prognosis of these disorders is better than would appear at first sight, the lingual development should be carefully improved by practice therapy. It will then often be found that the intellect is not disturbed, or only slightly, and has been underestimated owing to the lingual defect. This was also distinctly the case with the child I have mentioned. Of course, where imbecility has attained to a high degree, treatment of disorders of speech will encounter great difficulties.

21. DISORDERS OF SPEECH DUE TO AFFECTIONS OF THE PERIPHERAL NERVOUS SYSTEM AND THE MUSCULAR APPARATUS

DISORDERS OF SPEECH DUE TO PARALYSIS OF THE FACIAL

In spite of unilateral paralysis of the facial nerves, the labial sounds are usually correctly formed by distortion of the mouth, amounting to compensation of the affected side. This is exceptionally not the case when complications such as imbecility aggravate speech. In these cases the complicated labial sounds B, V, F, and M are absent, if there is only hemiparesis, while the purely explosive sound P can still be expressed. If the right facial nerve is chiefly attacked, Sh is also missing (protrusion of the lips). F is replaced by D or L, M by N, and there is also parasigmatism (see p. 402). Peyser observed unilateral clonic spasms of the velum with simultaneous partial paresis and open snuffing. In bilateral partial paralysis the sounds B and P are missing (Fig. 55, p. 381). They are replaced by labio-labial, weak friction sounds, while M is replaced by N. The labial sounds are completely absent in compound words. Compensatory phonation is gradually acquired by a strongly prognathous inferior maxilla, which will impart peculiar masticating movements to the act of speaking. Gutzmann mentions another form of compensation. The boy in question rapidly pressed his lower lips over the lower incisors with his fingers whenever he wanted to pronounce F. As the lip returned to its normal position, it grazed the upper teeth and at that moment the air current produced the labio-dental friction sound. The vowels, too, lose in distinctness by the absent movements of the orbicularis oris, although this defect can also be overcome by a vicarious movement of the fundus of the mouth. These characteristic disorders also occur alone at first in initial bulbar paralysis. Facial paralysis interfering with speech has also been observed in Basedow's disease.

As to speech disorders due to polyneuritis, especially of post-diphtherial palatal paralysis and aphonia, see Zappert, vol iv, p. 279. In myasthenia pseudoparalytica disturbances of speech may occur owing to rapid fatigue of the articulating musculature, causing unintelligible lallation with the characteristic manifestations of bulbar paralysis.

Up to the present only one case has been phonetically examined.

MUTISM AND APHASIA IN CHILDHOOD

Although these two affections have already been mentioned, they require a comprehensive survey on account of the differential diagnosis and their manifold etiology. The following division has been based principally upon the communications of W. Stern:

(1) **Mutitas Physiologica.**—Dumbness before beginning to speak should not go beyond the second or at the most the third year. (For further details, see *Lingual Development*, p. 361.)

(2) **Mutitas** (physiologica) **prolongata** and **audimutitas** (auditory dumbness) have been discussed on p. 388.

(3) **Mutitas** (physiologica) **prolongata** combined with, or caused by, (a) hardness of hearing (p. 380); (b) slight disturbance of intellect (p. 389); (c) defects of the peripheral organs of speech, as for instance the muscular articulation (p. 397) or of the palatal formation (p. 405); (d) the totality of these three complications (p. 442).

(4) **Mutitas idiotica**, the dumbness of imbeciles, with or without lingual understanding (p. 441).

(5) **Mutismus hystericus** (p. 438).

(6) **Aphasia**, being the loss of partly or completely acquired speech. (For "congenital" sensory aphasia, see p. 388.)

The following distinctions are made according to causes:

(a) **HYSTERICAL APHASIA**, described on p. 438.

(b) **SO-CALLED REFLEX APHASIA**, a form of speechlessness which might perhaps with good reason be classed with mutism, and in other cases with hysterical mutism.

Determining causes, as described in the extensive literature, are: Disorders of digestion, overeating (speech being restored by laxatives or emetics after having persisted for a week), helminthiasis (cured by appropriate therapy). Shock aphasia can only with difficulty be separated from hysterical conditions.

The case of a thirteen-year-old girl, mentioned by Wertner, was surely one of hysteria. She had been run over without sustaining any serious injuries; she was speechless for thirteen months and then declared she was ready to speak again. Demme reports the following case: Nine days' aphasia in a six-year-old girl after tenotomy of the tendo Achillis, normal speech restored on the twenty-first day after operation; this case should be classed under shock aphasia. There is, of course, no doubt that shock is liable to make people lose their voice.

(c) **APHASIA**, usually of the motor and rarely of the sensory form in and after infectious diseases, has often been observed in smallpox, measles (see Moser, vol. ii of this Handbook, p. 243), scarlet fever, influenza, but principally in typhoid fever. Clarus mentioned twelve cases of typhoid aphasia, ten of which were boys. The duration was three weeks. Henoch stated that the affection lasted from one to two weeks. Gutzmann says that true typhoid aphasia is rare (four cases), and refers to hysterical mutism in typhoid and soporose conditions. He, as well as Curschmann, believes that typhoid aphasia is as yet unexplained. In a large number of aphasia cases following infectious diseases there are encephalitic processes, often of a hemorrhagic character. This applies especially to influenza and pertussis.

Von Domarus, of F. von Mueller's Clinic, recently reported the

case of a six-year-old girl with febrile right-sided hemiplegia and aphasia occurring during recovery from a medium case of pertussis. (See also Neurath, vol. ii, p. 480.)

(d) **APHASIA** after injury to the head, indicating an involvement of the lingual centres, is very rare in childhood, simple concussion of the brain being apparently far more liable to destroy speech.

There is one of Duval's cases, however, which may serve as a classic example. After fracture of the frontal bone a boy lost his voice. Thirteen months later he was drowned. Autopsy findings: A cyst in the third left frontal convolution. Most of the other cases healed in a few weeks, which is quicker than in adults. Dinkler describes an exceptionally grave case. A child, two and one-half years old, remained psychically changed after the trauma, had paroxysmal headache, vertigo, vomiting and walking disturbances, but it was not until two and one-half years later that it began stuttering. Speech was rapidly lost and death occurred in coma. Autopsy revealed polio-encephalitis hemorrhagica inferior.

(e) **APHASIA IN OTHER ORGANIC AFFECTIONS OF THE BRAIN AND ITS MENINGES.**—Freud distinguishes retarded lingual development and true aphasia in infantile paralysis. The frequency of the latter, following hemiplegia, has been denied by Cotard and Féré. Bernhardt states that motor aphasia is not rare in childhood, but refers to all forms of aphasia of whatever etiology. Neither the age of the child nor the seat of the lesion, nor the extent and gravity of the paralysis in hemiplegia is a decisive factor in the arrest of speech (Wulff).

Freud has collected forty-four cases of aphasia, of which twenty-one were right-sided and twenty-one left-sided; furthermore, his table of two hundred cases of right paralysis includes eighty-nine cases of aphasia, and of two hundred cases of left paralysis, thirty-five cases of aphasia.

Negative symptoms do not often persist long, because evidently the right hemisphere easily vicariates for the left in childhood, and the localization is not firmly established. In a case of Pick, aphasia remained uncured, the lesion extending to both hemispheres. As a general rule, infantile cerebral paralysis causes speech to be restricted to a few words, like "yes," "no," "papa," "mamma," which are mechanically repeated while lingual understanding usually persists. In spite of the paralysis, speech will return from within a few weeks to a year, but the disorders described on p. 457 will persist for some time longer. Cure is therefore incomplete and independent of the behavior of the paralysis. The seat of tumors, should be present, has not the same significance as in adults. Clarus found in aphasia tumors in the right five times and nine times in the left cerebrum and three times in the cerebellum and pons (comp. Zappert, vol. iv, p. 236). So-called aphasia in chronic hydro-

scarcely is an index of mental impairment. In tuberculosis it is usually a symptom of cerebral. In the other hand pure aphasia occurs in meningitis, pneumonia. Sensory aphasia after infectious diseases may also be the consequence of subdural meningitis.

Acquired and hereditary aphasia seem to be very rare in childhood. In the subdural meningitis of the dura over the left frontal and parietal region it is important.

Acquired and sensory aphasia in epilepsy has been mentioned in § 483.

Stammering, inherited aphasia, is probably a form of anarthria, and not a disturbance with a mental cause (§ 485).

In clinical aphasia our intention is to assume the possibility of congenital aphasia. Nevertheless the term is used especially for those forms which owe their origin to diseases occurred at birth, just as the expression "congenital sensory aphasia" is used. Aside from epileptic disturbances, infantile aphasia usually takes a stammering course, terminating in more or less complete mutism. Its symptoms of stammering in childhood are characteristic. In the first place, both functional and organic aphasia are influenced by stammering, and in the second place they often terminate in so-called aphasic stammering. It does not seem justified, however to designate the muteness of such stammerers as stammering aphasia.

The prognosis of aphasia in childhood is generally good.

The object of the treatment is to train the non-paralyzed extremities. Writing exercises, even with the left hand, and exercises in articulation, as in stammering, bring good results which fully pay for the exerted trouble.

Aphasia voluntaria, or voluntary silence, is sometimes occasioned by a teacher.

For example, the historian, Goethe, as a seven-year-old boy suffering from epilepsy. Before entering school, he had had private lessons and when sent to a school, he got the stigma of the lingual defect. The impression was overwhelming. He came home weeping and refused to speak any more and he was persuaded that the defect could be easily removed. For example, as observed voluntary silence in a seven-year-old girl after an operation of tracheotomy, the child being perfectly aware of the operation and its result. The silence observed by hard stutters can persist. This stammering is caused by tonic spasms. The prognosis is not very favorable.

In such cases the following questions to be decided by the physician are: whether he can throw himself into the following: Whether he is conscious of the fact of uttering a sound; whether he is conscious of the fact of uttering a word; whether he is aphasic and cannot speak or whether he reserves voluntary silence.

IV. DISORDERS OF THE VOICE

1. PHYSIOLOGY OF THE INFANTILE VOICE

(1) **The voice of the nursling** has been analyzed by Flatau and Gutzmann in thirty cases. They found an astounding variation in the first reflex cry, considerable differences and anticipated motor complexes, the presence of which is of theoretical importance. The result of these investigations may therefore be briefly reviewed. The tones of preference usually lie in the triangular region between a medium and a soprano falsetto voice from a^1 - d^2 , sometimes going up to the double crossed octave, a^2 . The first cry is around a^1 and b^2 . Later, the range of the sounds comprises six to eight half tones. Above and outside these sounds many other characteristic, inspiratory tones will occur up to an octave higher, also falsetto and whistling sounds which may reach quite an inordinate height, up to d^4 . Thus, the voice of the examined infants ranges from g^1 - c^4 , but of course not with a continuous scale.

Among vowels, the \bar{a} -sound is the favorite, less often Ah , or alternations with I , E , A , O , U , which only plays an introductory part, as a rule. Whistling tones sound in E . Articulations, chiefly Ng , N , or, less often, V , occurred only in seven infants. The voice always sets in very firmly at first, but later becomes softer. There are, besides, a multitude of noises, interrupted vowel sounds, groaning sounds and cough-like explosions. A certain vacillating sequence of sounds is considered by these authors the "first palpable rhythmical formation." Gutzmann noticed the first "primitive song" in the fourteenth week (from e^1 to g^1). The whistling and inspiratory tones and elements of articulation gradually disappear, and the "song-like store is more and more reduced in favor of the development of speech." Gutzmann's little daughter made rudimentary singing attempts when eleven and twelve months old (d^1 - f^1 ; f -sharp¹- d^1), going up and down; then followed c -sharp¹ to g -sharp¹; below g^1 there was pectoral voice and above g^1 falsetto. For the cry respiration of infants see p. 360.

(2) **The infantile voice** is of particular importance to the physician on account of the great frequency of hoarseness among school children. The range of the speaking voice is very great between the third and seventh years. It comprises nine half tones (a - f -sharp¹) in both boys and girls; after exclusion of the very high and very deep sounds, which rarely occur, the voice ranges between g and e^1 according to Paulsen, between a and d^1 according to Gutzmann. The range of the singing voice is subject to considerable variations. According to more recent

investigations of Garbini, Paulsen, Flatau, and Gutzmann, it comprises five half tones in the first and second years, taking the most frequent tones into consideration and using real, not geometrical, averages; in the twelfth year it comprises fourteen to nineteen half tones in boys and sixteen to twenty-two half tones in girls, which will not materially increase up to the change of voice. The growth of the increase has been illustrated by Gutzmann (Fig. 74). It progresses less rapidly upward in boys, the range of boys' voices being smaller than that of girls. The lower limit of the infantile voice is on the average near g and a , the upper one near g^2 - b^2 . The range is greatest in the tenth and eleventh years, after which the upper limit sinks in both sexes. The very great difference in extent of children's voices is shown by figures furnished by Paulsen, Flatau, and Gutzmann. They found ten to thirty-one half tones in the seventh and sixteen to forty-one in the fourteenth year; the proportion of frequency between a great and a small range was 1 : 3. Exceptional cases will not be described here, as for instance a child of musical parents, observed by Flatau, who commanded five tones at the age of nine months, and two octaves at the age of four years, although possibly these cases are not particularly rare. Among the 575 children



examined by Flatau and Gutzmann, 3-7 per cent. were unable to "repeat any musical sounds at all in spite of a thoroughly normal condition of the organs," (so-called hummers). Imhofer mentions the considerably smaller range of voice in weak-minded children.

The range of voice is also of considerable importance for singing in school, to which further reference will be made.

Aside from this point, it is necessary, in the interests of voice formation, to know the register of children's voices. Here again Gutzmann and Flatau make detailed statements. Most children have a so-called principal register (also called the middle voice) which ranges from about b - f -sharp² and also serves the purposes of speaking.

The fissure of the glottis is narrow and long, there are broad curves at the vocal cords, the larynx occupies a middle position, and there is no pectoral resonance. The character of the vocal sound in this register determines the timbre of the infantile voice.

Below this border-line there is a pectoral register, ranging from D to A , which is characterized by palpable thoracic vibrations; this register, however, is not always present. The laryngeal picture resembles

in a general way the one described. Above the principal register, the falsetto voice comprises in the average case the sounds g -sharp²- d^3 . This register as well as the principal register have nothing in common with the falsetto voice of adults, with which they have been erroneously identified.

In producing falsetto sounds, the vocal cords become longer and narrower, the ventricular bands follow suit, and the glottis fissure itself becomes still finer and narrower.

Flatau describes a fourth, or whistling, register which is found in 5 per cent. of girls from about nine to thirteen years; in rare cases it will be preserved. The sounds of this register are above E^3 and of a metallic or flute-like character.

It was found in the few cases which have been carefully examined that the glottis usually assumes a fusiform shape (whistling fissure), while the soft palate is considerably drawn up. The velum is firmly pressed against the faucial wall, the palatal arches approach each other to the utmost, leaving but a narrow space between them, while the epiglottis descends very deeply (Fig. 75).

Most children possess a pectoral and a principal register, and a comparatively large number have falsetto sounds besides; it is only exceptionally that either of the registers exists alone.

The rarity of a falsetto register is remarkable and probably due to improper training. In boys, the pectoral tones gradually increase up to puberty.

(3) **Mutation of the voice** in both, girls and boys, is of the greatest importance for the further training of the voice. Any disturbance may cause permanent injury, and this is often confused with organic affections of the larynx.

In regard to development of the larynx at puberty, see Seitz, this Handbook, vol. iii, p. 111. The vocal cords in the male grow longer in a short time by one-third; any asymmetries which occur during this period will be compensated in the course of growth.

The voice of boys gradually sinks by an octave, that of girls only by a third. The speaking voice of boys suffers a corresponding change, going down to d or e in A .

According to Paulsen's investigations, 6 per cent. of the boys under fourteen spoke in adult tones, while 3 per cent. of those under nineteen still had the boys' voice. As a rule, the same voice is used for speaking and singing, but there are also cases where the adult voice is used for speaking and the juvenile voice for singing, or *vice versa*.

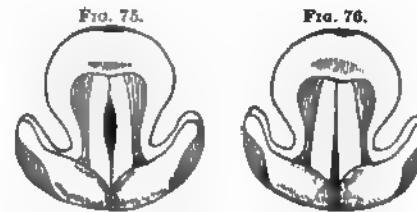


FIG. 75.—Diagram of the glottis fissure on sounding the whistling register.

FIG. 76.—Diagram of the glottis fissure in mutation.

Really striking disturbances of the voice at puberty (see Seitz, vol. ii, p. 111), consisting in pronounced hoarseness, aphonia and changing the voice either to falsetto or to abnormal depth, occur in boys oftener than in girls. As this condition is often accompanied by considerable hyperæmia and swelling of the vocal cords, it may be confused with laryngitis, causing useless application of a variety of remedies. Paulsen states that inability to sing increases in direct ratio from the thirteenth to the fifteenth year, where the climax is reached at 40.1 per cent.; it decreases in frequency from the fifteenth to the nineteenth year. Flatau, who has described the changes of mutation in detail, distinguishes the following:

(2) Persistent falsetto voice, in which the boyish timbre remains between a and e', or may possibly reach the top note of the adult voice, d or e. Gutzmann explains this by a preponderance of the cricothyroid muscle which assists the thyreo-arytenoid as abductor of the vocal cords, since with exaggerated length the latter fails to perform its function. The former muscle, however, produces the falsetto voice, and its functional insufficiency will easily change the voice to a deep bass, the vocal cords being slackened. This persistent wrong use of the falsetto voice causes spastic contractions of the external laryngeal musculature, followed by manifestations of fatigue due to unusual exercise, culminating in pain and pressure. This condition is accompanied by disturbed respiration in the shape of superficial, hasty respiration while speaking, and perverse action of the vocal cords in inspiration. The persistent use of the falsetto voice, therefore, resembles stuttering and occurs together with it. It also occurs in the families of stutterers.

(4) Perverse mutation in girls: The larynx undergoes exaggerated development, causing the voice to assume a rough and deep timbre. Gutzman mentions a girl of sixteen whose voice ranged from C-b², Gutzman a girl of thirteen whose voice ranged from a deep bass to the normal timbre of an adult woman. Flatau, on the other hand, describes arrest of development of the larynx in the male with very slight change of voice and underdevelopment of the sexual organs.

Premature low change of voice has been often observed as a symptom of *pubertas præcoca*. A child, described by S — with a deep voice. Neurath has compiled a

ternal Medicine and Pediatrics," vol. iv. He suspects tumors of the pineal gland, Lange (vol. iv, p. 9) tumors of the genital glands and adrenals.

The *treatment* of disorders of the voice at puberty should set in as soon as there is no further change and the gradual development of the normal voice stops, or if the prevailing hoarseness is felt as a serious disturbance. Singing at school should be discontinued. Practising in the correct timbre is usually sufficient for a cure. Should the falsetto persist, special practice is necessary, by commencing with breathing sounds, then whispering sounds, followed by practising single sounds in as low a key as possible. This is the same method as employed in stuttering, slight pressure on the thyroid cartilage being an aid to phonation at first. A combined application of the electric current, massage and compression has given excellent results in my hands. When changing from vowel to consonant, or *vice versa*, the same key should be retained; the voice should set in gently, not compressed, and expiration should be prolonged. Reading and reciting will bring the treatment to an end, which, in light cases, will occupy but a few weeks, while in persistent cases it may have to be continued for several months. Local treatment of the larynx is useless. The disorders in organic affections of the nervous system have been treated of on p. 451.

V. IMPORTANCE AND PROPHYLAXIS OF DISORDERS OF SPEECH AND VOICE IN SCHOOL

THE number of school children in Germany with disorders of speech and voice are about 200,000, as shown by numerous statistical figures compiled and published by Gutzmann. The mental development of a great number of these is necessarily backward and according to Sarbo they amount to 23 per cent.

Most of these children enter school without any adequate lingual development, others had previously suffered from lingual disorders, such as auditory mutism, pronounced spluttering, akataphasia and stammering, which have prevented their normal intellectual development; others are intellectually deficient by nature or have remained undeveloped by illness or neglect. All are behind their normal schoolmates of the same age. The first lessons remove stammering in a large number of cases, the number (102,000) being thus rapidly reduced (see Fig. 77). The rest of the stammerers are often accused of "speech-laziness," scolded in school instead of being sent to a specialist, frightened instead of cured. It would be a far better plan to free these children from their defects before entering school, and this can be accomplished between the ages of four and six. It devolves upon the family physician to call parents' attention to this fact instead of consoling them with the idea that the disorder will "disappear by itself." The foundation of kindergartens for these children, as has been accomplished by Knopf, in Frankfort-on-the-Main, should be more generally adopted. Although our public schools are generally well organized, so far as speaking and reading are concerned, the details of lingual education may well be improved

upon in many particulars, as may be seen from the following pages.

While stammering decreases in frequency during the first school years, the number of stutterers is rapidly on the increase. Statistical figures comprising half a million children in Germany justify an estimate of 98,000 stutterers among them, or 1 per cent. At the beginning of school age the number of stutterers amounts only to 0.5 per cent., while in the higher grades they have increased to 1.5 per cent. (see Fig. 78). A still greater increase of stutterers will be found in the private

FIG. 77.



This figure shows the percentage of stammerers of a certain age, the decrease of stammering during school age being very rapid.

schools, where exact statistics have shown a considerable increase at the ages of seven and eight. This, then, is immediately after the first lessons in reading and writing, the difficulties of which must be regarded as exciting causes. An important reason for this fact is, according to Gutzmann, that a relatively rapid period of cerebral growth terminates with the eighth year. (Compare Pfister, this Handbook, vol. iv, p. 121.) Another point is that slight lingual defects are discovered only at school. The demands of the first lessons, too strenuous methods, may cause the affection to break out in children with a family taint, and who are shy and easily frightened. Again, imitation of stuttering schoolmates is often a cause in early school years. The number of stutterers is somewhat reduced in the ninth year, while from twelve to fourteen it is again

considerably increased, probably under the influence of manifestations of puberty. This, however, only occurs in boys.

It is quite clear that a serious defect like stuttering, which is often associated with psychic injuries, must have an unfavorable effect upon progress in school.

Gutzmann estimates that 50 per cent. of stutterers are backward pupils, although on the average stutterers are not ungifted. According to Sarbo the percentage is only 36 per cent. (2208 out of 6046); the number of backward boys having been found to amount to 36 and that of backward girls to 37 per cent. Westergaard's statement that all stutterers sit in the lower benches has not been confirmed in my

experience. Lindenberg, who has compiled statistics of 212,000 school children, among whom there were 7.4-9 per cent. of stutterers, found that the number of bright children among the latter are in the minority.

Part of these children are purposely neglected or not called up by the teacher. According to my experience, this occurs in private schools even more frequently than in public schools, the reason probably being that the written exercises are sometimes overestimated in the latter. The pupils often leave school with very slight lingual attainment. They cannot express themselves, not because of their stuttering, but because they have had no opportunity to practise speaking. Stutterers are often wrongly judged in school, although at the present time the majority of teachers look upon this affliction as a disease, and not a bad habit. But they are often enough exposed to the ridicule of classmates. Liebmenn points out that stuttering pupils are often reproached and punished, because they are supposed not to have prepared their lessons, the teacher knowing that they stutter harder the worse they come prepared. Greater



The figure shows how many of the 100 stutterers of the older grade have dropped out. Steady increase of stutterers during school period (After Gutzmann.)

severity in such a case would not be unjustified, if it were not pedagogically wrong to adopt harsh treatment, under the influence of which the affliction grows worse. Again, these pupils may remain silent for fear of stumbling over difficult sounds which may be contained in the answer. The same fear may cause these children to replace difficult sounds by others, thereby giving the impression of uncertainty. The teacher may thereupon insist upon repetition of badly pronounced words, with the result that by this very practice the difficulty is increased. This may be accompanied by wrong instructions and reproaches, such as to take a deep breath, without giving the subject of correct breathing any thought, to use more effort and energy, by which fear is increased with voluntary spasms in every part of the respiratory and speaking musculature. Thus the command: "Speak slowly, without stuttering," will usually aggravate the condition, although it may be admitted that this may have the desired effect in a large number who belong to Class I (see p. 417). But it is not the teacher's business to decide how an individual case is to be treated.

P. Maas communicated a case showing what serious results the good intentions of teachers may have. When Maas sent a boy back to school, after having effected considerable improvement, the teacher observed the boy to slightly stutter on pronouncing the sound K. He then had this sound immediately practised, with the result that the boy was totally unable to bring the sound over his lips at all, until correct treatment reassured him. Occurrences of this kind are well known to every lingual specialist.

The treatment of cured or improved stuttering in schools is not an easy matter for the teacher. He should exercise great care not to intimidate. He should never test children for their ability to speak when they return to school. A test is always a psychic insult of first-class magnitude. These patients should be spared immediately after they have returned from treatment. The worst method is to call the child's attention to his failure, as this will positively cause either a relapse or permanent deterioration.

Again, it would be a mistake not to let these children speak at all. The distrust with which they are affronted and which they will soon learn to understand makes them displeased and shakes their knowledge. How, then, should such a convalescent be treated? In one point, inexorable severity should be exercised, but not toward the afflicted child, but toward his classmates for teasing him in spite of a request to stop it. The self-consciousness and confidence of the patient should be awakened, the praise of his progress may even be somewhat exaggerated, he should be treated with friendly consideration, overlooking slight mistakes and relapses. The teacher may aid in giving an expected answer by speaking

the beginning of the answer with the child. Questions should therefore be clothed in the character of an answer, because its repetition will facilitate the task. If then the good progress of the child is referred to again and again, better results will be attained than by encouragement to greater energy, which after all is not meant sincerely.

At the same time, teachers and school physicians should have a certain knowledge of the psychology, physiology, and pathology of speech, in order to be able to correctly advise the parents and to impart the lessons in a way calculated to remove the defects or prevent their occurrence. Teachers should therefore undergo adequate training in seminaries and colleges. The details of prophylaxis are explained later. Courses for the cure of lingual defects of school children are always a makeshift, and the establishment of special classes will remain a pious wish.

The increasing frequency of chronic hoarseness is less known than the frequency of lingual defects. The percentage amounts to 41.6 per cent., as will be seen from Fig. 79. The causative factors are in the main the following:

- (a) Singing in chorus, in which there is more shouting than singing;
- (b) Exaggeration of the demands made upon the vocal range by practising songs which do not consider the majority of the children (75 per cent.);
- (c) Singing at puberty, the first manifestations are apt to be overlooked;
- (d) "The school-speaking voice," setting in with a hard tone which, according to Paulsen, is "unnatural, very loud and high." He found it to be a third higher than the natural speech of children and rightly blames an unnatural way of speaking "as being distasteful to the children." He applies the same remarks to the "exaggerated accentuation of syllables."

In view of the demands made to-day upon the children's voice, Garbini may well exclaim: "*Povri bambini.*" We are confronted with the serious question whether the maltreatment of the voice is an indifferent matter and whether the duty is not incumbent upon us "to protect the juvenile voice in the first school years with care and competence from the ill treatment of intensity" (Flatau and Gutzmann).

The prophylaxis of the lingual and vocal disorders in the school presupposes sufficient preliminary training of the teachers in the physiology of the speaking and singing voice and of the language generally.

This foundation for correct speaking, or lingual hygiene, is also important for the health of the teacher. His speech and instructions should be phonetically correct, while treatment of lingual and vocal disorders should be relegated to the physician. The services of the latter will

be so much less required as the first instructions in speaking, reading and singing are correct. These correct instructions will "remove the majority of grave lingual defects which, untreated, will blast the entire future career of a child" (Gutzmann).

Kerschensteiner has introduced the principle in Munich to spare the child with reading just after entering school, unless he is able to speak phonetically correct. During the first two months, lingual instructions are given on the phonetic principle. The language of the little ones might already be trained in the kindergarten, if the teachers had the necessary understanding for the physiology of speech. Correct instruction in phonation trains the infantile organs of speech, and forms an excellent foundation for progressive instruction and for facilitating the principles of orthography.

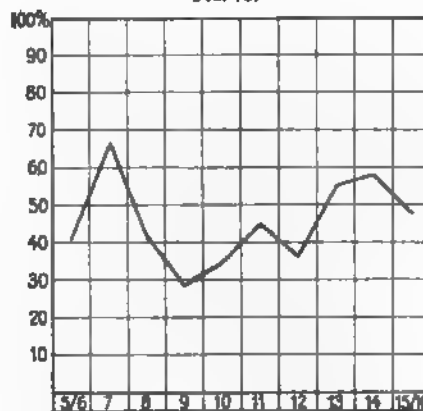
Diesterweg mentions as an additional advantage the time saved in learning to read, but Gutzmann rightly points out that it is far more important to have the children learn reading correctly than rapidly. Rapid learning promotes disorders of speech, careful learning prevents them.

Speech is acquired with the aid of the ear, the eye, and the tactile sense. The ear serves to compare the child's own language with the pure sounds of the teacher. However, he will learn from another child who speaks well already. There are physiological and psychological reasons why just in this period children should learn from each other. The eye serves to follow the position and movements of the organs of articulation, as practised by the teacher or a schoolmate, comparing them with his own in a mirror.

The use of the eyes in the first lesson, even with the aid of illustrations depicting the various positions, has also rendered excellent services in correcting light stammering defects. The tactile sense is used to control the setting in of the voice, the strength of the voice, the respiratory movements which are felt by the learning child when placing his hands against the larynx and chest of the teacher or speaking classmate. All these measures can be easily carried out in practice, and thus the various components consisting of respiration, voice, and articulation are practised separately and combined. This is the best prophylaxis against disorders of speech.

(1) Respiratory exercises are also of hygienic value; they should, therefore, not be confined to the first reading lessons, but also applied

FIG. 79.



Chronic hoarseness in 575 school children of both sexes, compiled according to percentage and age.

before and at later periods. They should be practised with closed mouth, if only hygienic exercise is intended; physiological speaking respiration, however, should be carried out rapidly, deeply and quite noiselessly through the mouth and with slow expiration (see p. 429). During inspiration and expiration there should be no sipping noise, no groaning due to approximation of the vocal cords, no explosion of breath, no unnecessary movements like raising the shoulders, keeping the head stiff, etc. In respiration through the nose, all snuffling sounds due to aspiration of the nostrils should be avoided. The movements are first observed by the child and then imitated before a mirror, with his hands placed upon his chest.

(2) Exercising the voice, including the vowels, should commence with whispering. This should be accompanied by exercising for positions, which are followed by a moderately loud, soft sound. Through the breathing sounds, the voice will be trained to set in softly; I consider it wrong and dangerous to specially practise the initial explosive sounds. It is used too often even without practice. The voice, even when practising vowel sounds, should be kept at a low pitch, because the normal speaking voice is at the lower end of the vocal scale. Duration of sounds and their modulation have to be specially practised, if a pleasant use of the speaking voice is to be acquired. Bad habits should therefore be prevented early by teaching the child to practise short open vowels and sounding consonants with distinct positions of the mouth and a low initial sound. This is at first practised by touching the laryngeal cartilage with the finger. In this way the exaggerated high, loud, and hard initial sound, pronounced with tense vocal lips, will be avoided; soft, moderately loud vowels, spoken in a deep key, will relax the vocal cords, which will be the best practice to prevent the development of chronic hoarseness and vowel stuttering.

(3) Articulating exercises can be practised for many sounds with the aid of a mirror. The expiratory current in explosive sounds can be illustrated by blowing a little card away, causing the mirror to become misty, etc. Media and tenuis should be carefully observed. Friction sounds and sounding consonants should be practised like long vowel sounds.

(4) The reading exercises should be based upon the same principles. The test of distinct whispering will show that it is not the high and loud voice, but careful articulation, which makes for intelligent speech. Appropriate distribution of the respiration over the entire sentence is of importance, care being taken that there will be no short, noisy inspirations between words or syllables. As the child reaches higher stages, correct, logical reading should be taught with elegant modulation and at a moderate speed. Strange to say, this demand is better fulfilled in public schools than in private schools.

If these rules are followed, doing away with the objectionable school tone and avoiding undue haste in answering questions, speech and voice can be so well trained in expression and modulation in schools that it will safeguard against the occurrence of disturbances.

(5) Singing in school, and especially singing in chorus, urgently demands reform in the interests of saving and training the voice. There is a consensus of opinion among all experts, from Manuel Garcia to the present day, in this respect. To effect this, however, singing teachers should know the exact range of the majority of the pupils. They should remember that most songs require a range of tones which no more than about 25 per cent. of children can command. They should know, and duly consider, the manifestations of puberty in both boys and girls, even in the absence of real vocal disturbances. Girls, at the time of the first menstruation, should not sing on any account. Chorus singing should be prohibited in kindergartens; on other occasions it should be replaced by solo songs or singing in small groups, which would obviate overexertion both as to length of time and loudness. Chronic hoarseness of school children and the grave voice disturbances at puberty would be most effectually overcome without curtailing singing in school, the educational and hygienic value of which is not to be underrated. This might also be the means of checking the evident decrease of good vocalists.

Prophylaxis of voice and speech disturbances in the school demands extensive physiological and pedagogic knowledge, and, although the task is an arduous one, it is thankful for school physicians, family physicians, and teachers alike.

VI. THE INABILITY TO LEARN TO READ. SO-CALLED CONGENITAL WORD-BLINDNESS

Definition.—The inability to learn to read deals with a manifestation wherein an otherwise intelligent or apparently intelligent child, who, while able to speak, draw figures, is well informed in general and able to memorize well, acquires the ability to read and to do spontaneous writing only with great difficulty, very incompletely, or not at all. Based upon my observations and on descriptions in the literature, the symptoms of so-called "Congenital Word-blindness" are as follows:

Symptomatology.—Most, but not all, of these children are intelligent—sometimes they even are among the better pupils. Often they have a good acoustic memory and may be able to recite pieces from their reader from memory, and by so doing deceive their teachers about their inability to read. Most of them also draw well and have also a good "optic memory." They are therefore described as "Optic Perception Types" (among others by Thomas), and their acute sense of observation is remarkable (Otto Wernicke).

They compute to the extent of their normal powers quite well; read figures and letters well; but learn particularly the last only with great effort, and sometimes those letters which are used less often not at all.

Words they cannot always read, even if in their own handwriting, or they learn with difficulty to read small words, for instance their own name, the names of the days of the week and the months. When a little advanced their favorite mistakes in reading are "Mistakes of Adding." In severer cases this becomes complete nonsense; for instance, they read instead of "But how do you know this" : "But now go—know with" (case of Thomas). Words spelled to them they cannot put together and therefore cannot pronounce them (Rutherford, McCall).

Copying is nearly always a retracing without understanding. Self-writing and dictation writing show the same mistakes as their reading. They write their own name as a picture (Siegel), without spelling. It also happens that they interpret matter, wrongly read, as perfectly correct (Variot and Lecomte). The reading of musical notes was impossible in several cases examined (Kilmer, Thomas), confined to one staff like violin music, but inadequate for piano music (Plate).

Disturbances of Speech.—Little is recorded of disturbances of speech as shown in two of my cases. However, Kerr mentions "Dysarthric Speech"; Förster, "Lisping Speech"; Variot, "Retarded Speech

Development"; and Thomas and Fischer speak of "Disturbances of Speech in the Family." Other authors emphasize good speech and mastery of several languages.

Course.—The inability to read seems to gradually disappear in most cases, but some defect always remains. A boy fond of sports finally learns to read the football scores, whereas in school he could never read (Hinshelwood). Military writers and natural scientists have their work corrected on account of the peculiar mistakes in writing (Plate). One "word-blind" case became a lawyer. Occasionally the disturbance remains uncorrected for lack of proper teaching, as in a case reported by Rieger.

Prevalence.—Including my own, I can so far find sixty-four cases in literature, mostly in school age, forty-seven males, seventeen females. The condition was first described by James Kerr in 1896. A case of Gustav Wolff observed in 1895 and published in 1902 concerns a weak-minded alcoholic. There is so far only one statement about the frequency of these cases by Warburg, who found among 2000 public school children, fourteen, and among 474* "Auxiliary School" children, fourteen to be "word-blind."

The disease is frequently common in the same family; as many as six cases have been observed in one family (Plate, Thomas, Hinshelwood). It is more frequent among the weak-minded, part manifestation of a disturbed intelligence.

Designation.—Rieger called this disturbance "partial idiocy in the reading of coherent words alone and also of numbers," and declines to use the expression "word-blind" for good reasons—because he wants the component "blind" used only in connection with conditions in which vision is abolished. Dejerine would like to have the term "word-blind" used only for an acquired condition (aphasia), but not for congenital cases. But since the "aphasia doctrine" has entered—and not in a very happy way—into the doctrine of the so-called congenital "word-blindness," it will be advantageous to follow Rieger in dropping this designation and to call it "Inability to Read" (Nettleship).

Etiology.—In explanation of this disturbance most authors, with the exception of Rieger and his pupil Reichardt, deduce the same from a hypothetic defect in the gyrus angularis in shape of hemorrhage due to trauma during birth, or a congenital hypoplasia of this region (Fisher), or defects of the commissure (Bastian). Rutherford considers it the loss or destruction of a highly differentiated area in the primitive streak. His cases occurred at the end of a long row of successive births (British Journal of Children's Diseases, Nov., 1909). He sees in them a kind of

* "*Hilfsschule*" is the name of a private school in Germany which takes the place of our "tutors" and "props."

atavism, a falling back to a lower type of intelligence. The existence of a special reading centre is hypothetical. Wernicke himself denies its existence, as do his pupils Sachs and others, while Dejerine accepts it. Since the reading act takes place by way of Wernicke's centre, we may accept that in general we read by way of our sensory speech centre, which is proved by the research in the psychology of reading. The acceptance of a mechanical disturbance of the association tracts between the centre of vision and of hearing in such cases of inability to learn to read was also considered. Gross anatomic changes have never been observed. The course of most of these cases also speaks against such theory. Nevertheless, in such cases, upon this hypothesis, it has been suggested to practise writing with the left hand, just as in the treatment of motor aphasia. Nothing is published about the success of such experiments.

Based upon our knowledge of the psychology of reading of the child and adult, it is possible to explain these disturbances in two ways, and further research will have to prove whether or not there are various types, which from dissimilar causes give the same train of symptoms. It may be that it is a defective associative connection between Word (letter) picture and Motoric (articulated) word, but it may also be that a word picture as such is not at all or only incompletely apperceived, a disturbance in the apperception of quality of form or (Letter-picture). Whatever it be, the real cause must be decided by future analysis.

The proof of remarkable weakness in the retaining of sound associations and the ability of immediate apperception of succession of words as given by Ritter further the "inability to give the pronunciation of words, the letters of which were spelled over."

As observed by Rutherford, furthermore, the peculiar forgetfulness for commissions in spite of normal ability to memorize as emphasized by Warburg, and finally retardation in the development of speech (Nadoleczny), will all furnish material for a psychologic analysis.

Treatment.—Such a careful psychologic analysis of each individual case is absolutely necessary for rational treatment. It will be possible to decide then which method of tutoring, analytic or synthetic methods of teaching to read (Phonic System or Look and Say Methods, C. T. Thomas) deserves preference. The first will improve visual perception but not auditive, as Hinshelwood points out. The method of teaching to read as practised in Germany will be the best in general for all cases not of an extreme "visual perception" type.

The results of such exact method which considers the peculiarities of this disturbance are in general satisfactory, according to reports of authors of extended experience, in spite of the fact that a certain inability to read can always be detected.

LITERATURE

No complete bibliography has yet been compiled.

1. James Kerr: School Hygiene in its Mental, Moral and Physical Aspects. The Howard medal prize essay, 1896.
2. James Kerr: *Lancet*, May 19, 1900, p. 1446.
3. James Kerr: Annual report of the medical officer of the late school board for London for 1904, pp. 30-33.
4. James Kerr: Physiological Variations in Children. *Child Study*, October, 1909.
5. James Hinshelwood: The Visual Memory for Words and Figures. *Brit. Med. Jour.*, Nov. 21, 1896, p. 1543.
6. James Hinshelwood: Congenital Word-blindness. *Lancet*, May 26, 1900.
7. James Hinshelwood: Congenital Word-blindness. *Ophthalmic Review*, April, 1902.
8. James Hinshelwood: A Case of Congenital Word-blindness. *Brit. Med. Jour.*, Nov. 12, 1904, and *Ophthalmoscope*, 1904, p. 399.
9. James Hinshelwood: Four Cases of Congenital Word-blindness, Occurring in the Same Family. *Brit. Med. Jour.*, Nov. 2, 1907, p. 1229.
10. James Hinshelwood: Two Cases of Congenital Word-blindness. *Brit. Med. Jour.*, March 18, 1911, p. 608.
11. James Hinshelwood: The Treatment of Word-blindness, Acquired and Congenital. *Brit. Med. Jour.*, Oct. 19, 1912.
12. W. Pringle Morgan: A Case of Congenital Word-blindness. *Brit. Med. Jour.*, Nov. 7, 1896, p. 1378.
13. Nettleship: Cases of Congenital Word-blindness (inability to learn to read). *The Ophthalmic Review*, 1901.
14. C. Ritter: Unfähigkeit zu lesen und Diktat zu schreiben, bei Voller Sprechfähigkeit. *Zeitschr. f. Psychologie u. Physiologie der Sinnesorgane*, 1902, B. 28, p. 96.
15. Gustav Wolff: Zur Pathologie des Lesens und Schreibens. *Allgem. Zeitschr. f. Psychiatrie*, vol. 60, fasc. 4, p. 509, 1903.
16. Otto Wernicke: Angeborene Wortblindheit. *Zentralblatt für Augenheilkunde*, 1903, p. 264.
17. Lechner: Aangeboren Woord-blindheid. *Weekblad van het Nederl. Tijdschrift voor Geneeskunde*, 1903, Nr. 5.
18. Stephenson: Congenital Word-blindness. *The Lancet*, Sept. 17, 1904, p. 827.
19. Stephenson: Six Cases of Congenital Word-blindness, Affecting Three Generations of One Family. *Ophthalmoscope*, Sept., 1907, p. 482.
20. Brunner: Congenital Word-blindness. *Ophthalmology*, 1905; ref. in *Ophthalmoscope*, 1905, p. 184.
21. Förster: Kongenitale Wort-blindheit bei Einem Schwachsinnigen. *Soc. de Neurologie de Paris*, 4 fevr., 1904; ref. *Neurol. Zentralblatt*, 1905, p. 235.
22. C. J. Thomas: Congenital Word-blindness and Its Treatment. *Ophthalmic Review*, August, 1905; *Ophthalmoscope*, 1905, p. 380.
23. C. J. Thomas: The Aphasias of Childhood and Educational Hygiene. *Public Health*, May, 1908.
24. Fisher: Case of Congenital Word-blindness. *Ophthalm. Review*, Nov., 1905, p. 315.
25. Fisher: Congenital Word-blindness. *The Ophthalmoscope*, 1911, p. 207.
26. Batten: Diskussion zu Fisher. (23.)
27. Variot et Lecomte: Un Cas de Typhlolexie Congénitale. *Gazette des Hôpitaux*, Nr. 124, Oct. 30, 1906.
28. Clairbone: Types of Congenital Symbol Amblyopia. *The Jour. of the Am. Med. Assoc.*, Dec. 1, 1906.
29. A. Schapring: a. Congenital Word-blindness in Pupils of Public Schools; b. On Two Cases of Word-blindness. *New York Academie of Medicine*, Feb. 19, 1906. Sections of *Ophthalmology*.
30. Peters: Ueber Kongenitale Wort-blindheit. *München. med. Wochenschrift*, 1908, Nr. 21.
31. Rieger: Ueber Apparate in dem Hirn. *Arbeiten aus der Psychiatr. Klinik zu Würzburg*, Jena, 1903, p. 47.

32. Plate: Vier Fälle von Kongenitaler Wort-blindheit in Einer Familie. Münchener med. Wochenschrift, 1909, Nr. 35.
33. Rutherford: The Ætiology of Congenital Word-blindness: with an Example. Brit. Jour. of Children's Diseases, Nov., 1909; ref. Ophthalmoscope, 1910, p. 30.
34. Warburg: Ueber die Angeborene Wort-blindheit und die Bedeutung Ihrer Kenntnis für den Unterricht. Beiträge zur Kinderforschung und Heilerziehung, Heft 79. Langensalza, 1911.
35. Pritchard: Intermittent Word-blindness. The Ophthalmoscope, 1911, p. 171.
36. Schröck: Ueber Kongenitale Wort-blindheit. Dissertation, Rostock, 1912.
37. Bastian: Aphasia and Other Speech Diseases, London, 1898.
38. Reichardt: Ueber Umschriebene Defekte bei Idioten und Normalen. Verhandlungen des Vereins Bayer. Psychiatr., 1908. Zeitschr. f. Psychiatrie, B. 66, S. 157.
39. Elliot Square: Discussion zu Hinshelwood, Nr. 9.
40. Cyrill Walther: Discussion zu Hinshelwood, Nr. 9.
41. W. Kilmer: Discussion zu Clairbone, Nr. 28.
42. Eva McCall: Two Cases of Congenital Aphasia in Children. Brit. Med. Jour., May 13, 1911, p. 1105.
43. Theodor Heller: Heilpädagogik, II. Edition, 1912, p. 153.
44. Nadoleczny: Ueber die Unfähigkeit Lesen zu Lernen (sogenannte Kongenitale Wort-blindheit) und Ihre Beziehung zu den Sprachstörungen. Münchener Gesellschaft für Kinderheilkunde, Feb. 21, 1913.

INDEX

PART I

DISEASES OF THE EYE *

	PAGE		PAGE
Abscess, lachrymal.....	11	Albuminuric retinitis.....	275
Absence of a fovea centralis.....	36	differential diagnosis.....	277
of Bell's phenomenon upon closure		prognosis.....	278
of the lid.....	43	treatment of.....	278
Accommodation, anomalies of.....	285	Amaurosis.....	275
disturbances of.....	300	Amaurotic pupillary rigidity.....	307
treatment of.....	303	Amblyopia.....	4, 268
of infants' eyes.....	62	congenital.....	36
neurasthenic weakness of.....	302	ex anopsia.....	27
paralysis of.....	301, 302	toxic.....	274
as a late manifestation of		Amyloid tumor.....	85
syphilis.....	303	Anæmia, chronic.....	187
relative range of.....	301	general.....	126
symptomatic weakness of.....	302	Anæsthesia, general.....	66
Accommodative spasm.....	294	local.....	64
Acne varioliformis.....	94	Anæsthesin.....	131
treatment of.....	95	Angiobлеpharon.....	8
Acquired alopecia of the cilia.....	97	Angiomas.....	6
Actinomycosis.....	87	Angiomata of the bulbar conjunctiva.....	11
diagnosis of.....	87	Angiosarcoma.....	283
treatment of.....	87	Aniridia.....	16, 19
Acute dacryocystitis.....	118	Anisometropia.....	4
and blennorrhœa of the lach-		Ankyloblepharon acquis.....	105
rymal sac.....	118	Annular keratitis.....	213
diagnosis of.....	119	ulcers.....	200
metastatic suppurative iridocho-		Anomalies, congenital, of the cornea.....	14
rioiditis.....	252	of the conjunctiva, congenital.....	11
treatment of.....	253	of refraction and accommodation.....	285
ophthalmogonorrhœa.....	155	Anophthalmos.....	42
pemphigus.....	97	with orbitopalpebral cysts.....	39
syphilitic tarsitis.....	86	Anterior polar cataract.....	25
treatment of.....	86	synechiæ, congenital.....	20
Addison's disease.....	126	Anthrax infection of the lids.....	77
Adipose or atonic ptosis.....	43	prognosis of.....	78
Affections of the conjunctiva.....	125	treatment of.....	78
of the cornea.....	191	Aplasia of the optic nerve and retina.....	38
of the external muscles of the eye.....	310	Areolar and disseminated chorioiditis.....	212
of the eyelids.....	67	Argyll-Robertson's sign.....	307
of the lachrymal organs.....	110	Artery, hyaloid, persistence of the	
of the lens.....	257	posterior part.....	37
of the levator palpebræ.....	107	persistent.....	19
ocular, due to ancestry, heredity,		persisting.....	31
or consanguinity.....	44	Artificial eye.....	253
of the optic nerve and retina.....	263	Asthenopia.....	310
of the sclera.....	225	treatment of.....	310
Albinism.....	4, 19, 40, 44		

* For index of disorders of speech, see page 495.

	PAGE		PAGE
Astigmatism.....	298	Caruncle, dermoids of.....	13
corneal.....	4, 14	Cataract.....	4, 14
irregular.....	300	anterior central, treatment of.....	30
treatment of.....	300	polar.....	25
Atonic ptosis.....	43	calcareous.....	259
Atresia, membranous.....	10	capsular.....	257
of the puncta lacrimalia.....	9	central, anterior.....	30
Auto-suggestion.....	298	circular.....	30
		congenital.....	4, 19, 24
Bacteriological findings in acute con-		fetal, treatment of.....	25
junctivitis.....	128	"Coppock,".....	25
Bandage, duplex.....	220	hereditary.....	25
Barlow's disease.....	69, 233	laminated.....	24, 258
course of.....	233	partial stationary.....	26
subperiosteal hemorrhages in.....	329	polar, treatment of.....	28
Basedow's disease.....	14, 69	posterior polar.....	23, 25, 257
Bilateral blindness.....	307	total.....	30
metastatic ophthalmia.....	252	treatment of.....	30
ptosis.....	5	Catarrh of the conjunctiva.....	127
Blennorrhœa.....	9, 118	conjunctival.....	180
conjunctival.....	10	follicular.....	126, 178
differential diagnosis of.....	10	spring.....	126, 174, 184
treatment of.....	11	differential diagnosis of.....	185
of the lachrymal canals.....	118	treatment of.....	185
treatment of.....	118	Catarrhal conjunctivitis, chronic.....	135
of the lachrymal sac.....	118	of the cornea.....	104
neonatorum.....	79	prognosis of.....	157, 158
Blepharitis angularis.....	135	Catarrhus siccus.....	135
Blepharochalasis.....	43	Cat's eye, congenital.....	20
Blepharophimosis.....	8, 102, 105	Central visual acuity.....	286
treatment of.....	105	Cerebrospinal meningitis.....	129
Blepharospasm.....	68, 99, 144	epidemic.....	271
inflammatory.....	101	Chalazion.....	85, 95, 183
prognosis of.....	99	treatment of.....	96
treatment of.....	99	Chancre of the lids.....	83
Blindness, bilateral.....	307	soft.....	86
causes of.....	47	Changes of the optic nerve in oxyce-	
congenital.....	46	phalia.....	280
Blood-pressure, transitory elevation of.....	126	Chemosis.....	188
Bovine conjunctival tuberculosis.....	183	filtration.....	188
Bulb, dermoids of.....	4	hyperæmic.....	188
Bulbar conjunctiva, angiomata of.....	11	serous.....	188
congenital teratoid osteoma of.....	13	Chlorosis.....	14
Buphthalmos.....	4	Choked disc.....	279
Burns and corrosions of the conjunctiva		differential diagnosis.....	279
and cornea.....	345	incipient.....	36
		treatment of.....	280
Canal, nasolachrymal, osseous, tuber-		Cholera.....	129
culous caries of.....	120	Chorioid, coloboma of.....	16, 30
Canaliculi, granuloma in.....	118	Chorioretinitis.....	4
Canals, lachrymal, blennorrhœa of.....	118	Chronic cyclitis.....	41
treatment of.....	118	Chronic dacryo-adenitis.....	112
Canthoplasty.....	102	Cicatricial contraction.....	106
Capsular cataract.....	257	deformities.....	78
Capsule, dissection of.....	26	entropion.....	105

	PAGE
Cilia, acquired alopecia of.....	97
Ciliary nerves, neurofibroma of.....	33
Cilioretinal vessels.....	35
Circulatory disturbances and hemor- rhages.....	187
treatment of.....	187
Circumscribed pathological pigment spots of the sclera.....	41
poliosis.....	40
Closure of the lid, absence of.....	68
defective.....	68
Coloboma.....	33
treatment of.....	6
of the choroid.....	16, 30
treatment of.....	30
congenital.....	5, 258
of the iris.....	5, 14, 16, 30
treatment of.....	30
of the lens.....	27
macular.....	18
of the optic nerve.....	37
palpebral.....	4
uveal.....	6
Color-blindness.....	44
"Color-hearing".....	38
Color-sense, of infants.....	60
Compresses, cold.....	134
warm chamomile.....	219
Concomitant and paralytic strabismus, difference between.....	312
squint.....	311
treatment of.....	315
strabismus.....	4
"Congenital amblyopia".....	47
anomalies of the conjunctiva.....	11
of the cornea.....	14
and deformities of the optic nerve and retina.....	35
of the lens.....	23
of the orbit.....	38
of pigmentation.....	40
of the uveal tract.....	16
of the vitreous.....	31
blindness.....	46
cataract.....	19, 24
changes of the sclera.....	22
defects of the eye.....	1
of motility of the ocular muscles.....	43
dermoids.....	12
disorders of the ocular nerves and muscles.....	41
ectropium uvulae.....	21
epithelial xerosis.....	12
exophthalmos.....	38

	PAGE
Congenital hemeralopia.....	266
occlusion of the lachrymal sac.....	10
opacities of the cornea.....	14
serous orbital cysts.....	40
staphyloma.....	15
teratoid osteoma of the bulbar con- junctiva.....	13
total or partial absence of the iris.....	19
Conjunctiva, affections of.....	125
diagnosis of.....	130
differential diagnosis.....	149
treatment of.....	130, 139
bulbar, angiomata of.....	11
congenital teratoid of.....	13
burns and corrosions of.....	345
treatment of.....	345
catarrh of.....	127
congenital anomalies of.....	11
croup of.....	146
diphtheria of.....	146
epithelial xerosis of.....	186
treatment of.....	187
extravasation of serum under.....	188
foreign bodies in.....	331
hyperæmia of.....	127
leprous affections of the.....	184
lipodermoids of.....	12
lymphoma of the.....	186
treatment of.....	186
new growths of.....	189
treatment of.....	189
non-pigmented nævi of.....	12
normal.....	125
orbital, gumma of.....	181
papilloma of the.....	189
treatment of.....	190
syphilitic affections of.....	179
diagnosis of.....	182
differential diagnosis.....	179
tuberculosis of.....	182
differential diagnosis of.....	183
treatment of.....	183, 184
variolar pustules of.....	145
treatment of.....	146
Conjunctival blennorrhœa.....	10
differential diagnosis of.....	10
treatment of.....	11
catarrh.....	180
folliculosis.....	178
xerosis.....	203
Conjunctivitis.....	70, 129, 131
acute, bacteriological findings in...	128
catarrhal, chronic.....	135
prognosis of.....	157, 158
treatment of.....	136

	PAGE		PAGE
Conjunctivitis, croupous.....	9, 147, 149	Croupous conjunctivitis.....	9, 147, 149
treatment of.....	149	treatment of.....	149
due to Koch-Weeks's bacillus.....	128	Cryptophthalmia.....	7
exanthematous.....	149	Cryptophthalmos.....	4
follicular.....	173	Cup, optic.....	1
gonoblenorrhœal, infantile, acute,		Cuts, stabs, and tears of the eyeball...	347
treatment of.....	166	Cyanosis of the retina.....	36
gonorrhœal.....	10	Cyclitis, chronic.....	41
gonorrhœal, abortive, metastatic...	168	Cyclopia.....	40
granular.....	149	Cycloplegics.....	301
granulosa.....	169	Cystic dilatation of the sac.....	114
of the Koch-Weeks's bacillus.....	135	Cysticercus, encapsulated.....	32
miliary phlyctenular.....	138	Cysts.....	109
plague.....	129	dermoid.....	6
pneumococcus.....	149	of the orbit.....	38
pustular.....	138	hydatoid.....	189
Connective tissue, vascularized.....	32	of the lower lid.....	19
Contusions of the eye.....	349	lymph.....	189
Convergents and divergens.....	311	orbital.....	19
Convergent contraction of the pupils...	306	congenital serous.....	40
Copper.....	340	orbitopalpebral.....	39
Corectopia.....	6, 21, 30, 33	scleral, congenital.....	22
treatment of.....	30	serous epithelial.....	189
Cornea, affections of.....	190	of the iris.....	20
burns and corrosions of.....	345	subconjunctival serous retention...	189
treatment of.....	345	traumatic.....	20, 189
catarrhal conjunctivitis and xerosis		Dacryo-adenitis.....	70
of.....	104	chronic.....	112
congenital anomalies of.....	14	Dacryocystic blennorrhœa.....	89
opacity of.....	4, 14	Dacryocystitis.....	70
eczematous ulcers of.....	198	acute.....	118
treatment of.....	199	diagnosis of.....	119
foreign bodies in.....	331, 334	of the lachrymal sac.....	118
herpes of.....	194	treatment of.....	121
in infants.....	50	chronic.....	119
marantic necrosis of.....	203	phlegmonous.....	10
treatment of.....	204	Dacryocysto-blennorrhœa.....	119
opacities of.....	6, 192	Darrier's disease.....	97
pannus of.....	171, 202	Deafness, reflex.....	307
differential diagnosis.....	202	Defective dentition.....	215
scrofulous processes of.....	202	Deformities, congenital, of the optic	
serpiginous ulcer of.....	207	nerve.....	35
staphyloma of.....	14	of the eye.....	4
tuberculosis of.....	215	Degeneration, signs of.....	5
Corneal astigmatism.....	4, 14	Dengue.....	129
changes in leprosy.....	223	Dentition, defective.....	215
treatment of.....	223	difficult.....	129
irregularities.....	191	Dermoid cysts.....	6
layers, foreign bodies in.....	192	of the orbit.....	38
leucoma.....	206	folds.....	13
opacities.....	192	Dermoids.....	15
origin of.....	192	of the bulb.....	4
Coryza.....	129	of the caruncle.....	13
Credé's prophylactic method.....	158, 159	congenital.....	12
Croup of the conjunctiva.....	146		

	PAGE		PAGE
Dermoids, epibulbar.....	19	Eczema, seborrhœica.....	89
treatment of.....	7	wet.....	91
Descemet's membrane.....	14, 15, 34	Eczematous blepharitis.....	92
Detachment of the retina.....	294	conjunctivitis.....	136
Development of the eye.....	1	ulcers of the cornea.....	198
of the infantile eye.....	49	treatment of.....	199
Diabetes.....	302	Elephantiasis mollis of the lids.....	33
Diagnosis of vaccine infection of the		Embryotoxon.....	14
lids.....	75	Encapsulated cysticercus.....	32
Diet in scrofula.....	141	Encephaly.....	42
Diffuse corneal infiltrate.....	211	Enophthalmus.....	39
Dilatation of the sac.....	114	Entropion.....	8, 105
Diphtheria of the conjunctiva.....	146	cicatricial.....	105
Disc, choked.....	279	treatment of.....	105
incipient.....	36	Epibulbar dermoids.....	19
optic.....	263	Epicanthus, congenital.....	7
Dissection of the capsule.....	26	Epiphora.....	113, 114, 116
Diseases of the orbit.....	321	labyrinthal.....	114
of the uveal tract.....	230	treatment of.....	115
Displacement of the iris.....	230	Episcleritis.....	226
of the lenticular nucleus.....	23	Epitarsus.....	8
Distichiasis.....	97, 105	Epithelial xerosis of the conjunctiva..	186
congenita.....	8	treatment of.....	187
treatment of.....	97	Epithelioma contagiosum.....	109
Disturbances of accommodation.....	300	Eruptive forms of syphilitic affections..	83
of pupillary movements.....	305	Erysipelas of the lids.....	76
treatment of.....	309	differential diagnosis.....	76
visual.....	263	treatment of.....	76
Divergens.....	311	Ewetzky's fold.....	13
Drops, instillation of.....	132	Examination of children with ocular	
Duct, lachrymal.....	3	affections.....	63
congenital anomalies of.....	9	of the pupillary function.....	305
strictures in.....	117	Exanthematous conjunctivitis.....	149
nasolachrymal.....	110	External injuries of the eye.....	343
anomalies of.....	110	treatment of.....	344
tuberculosis of.....	120	Exophthalmos.....	68
Duplex bandage.....	220	congenital.....	38
Dye, deformities of.....	4	pulsating.....	355
Ectasia.....	23	Extravasation of serum under the con-	
congenital.....	258	junctiva.....	188
Ectopia.....	23	Eye, artificial.....	253
of the lens.....	33	congenital defects of.....	1
pupillæ.....	21	contusions of.....	349
treatment of.....	23	development of.....	1
Ectropion.....	30, 68, 78	external injuries of.....	343
treatment of.....	30, 106	treatment of.....	344
Ectropium spasticum.....	106	infantile, development of.....	47
uxurians.....	106	injuries of the.....	331
Eczema, impetiginous.....	79	interior of, foreign bodies in.....	339
intrageminousum.....	89	massage of.....	221
of the lids.....	89	Eyeball, cuts, stabs, and tears of..	347, 349
prognosis of.....	90	Eyelids, affections of.....	67
treatment of.....	90	congenital pterygium of.....	8
pustulous.....	93	treatment of.....	9
		treatment of vaccine infection of..	75

	PAGE		PAGE
Facial clefts.....	19	Gonorrhœal conjunctivitis.....	10
nerve, paralysis of.....	68	ophthalmia.....	153
Faulty position of the cilia.....	97	Graefe's sign.....	108
Feeding in scrofula.....	141	symptom.....	69
Fetal trabeculæ.....	20	test.....	310
Fibrosarcoma.....	283	Granular conjunctivitis.....	149
Filtration chemosis.....	188	Granuloma in the canaliculi.....	118
Fissures, palpebral, gaping of.....	69	Granulomata.....	283
Fistulæ, lachrymal.....	9, 11	Grooves, optic.....	1
Flattening of the lids.....	67	Gumma of the iris.....	237
Follicular catarrh.....	178	of the orbital conjunctiva.....	181
conjunctivitis.....	173	Gummatous syphilides.....	84
Folliculitis ciliaris.....	95	syphilitic tarsitis, in hereditary or	
Foreign bodies in the cornea.....	334	acquired syphilis.....	85
in corneal layers.....	192		
in the conjunctiva, lachrymal		Habitual twitching of the lids.....	99
passages and cornea.....	331	Hæmangioma cavernosum.....	189
in the interior of the eye.....	339	treatment of.....	189
prognosis of.....	340	tuberosum simplex.....	109
treatment of.....	341	Hæmiatrophia facialis congenita.....	8
Fornicoblepharon.....	8	Hemeralopia.....	248
Fovea centralis, absence of.....	36	congenital.....	266
in infants.....	51	treatment of.....	248
Functional tests.....	63	Hemianopic reflex deafness.....	306
Fungous concretions.....	115	Hemophilia.....	69
Furuncles of the lids.....	80	Hemorrhages.....	187
		in the anterior chamber.....	233
Gangrene of the lids.....	78	orbital.....	125, 329
treatment of.....	79	treatment of.....	329
metastatic.....	78	recurring vitreous, treatment of..	251
Gaping of the palpebral fissures.....	69	spontaneous, and circulatory dis-	
Germinal vesicle.....	1	ease of the lids.....	69
Gland, lachrymal.....	3, 110	subconjunctival.....	187
inflammation of.....	111	treatment of.....	188
diagnosis of.....	111	subperiosteal, in Barlow's disease..	329
treatment of.....	112	vitreous, recurring.....	251
tuberculosis of.....	112	Hemorrhagic pemphigus.....	97
Glanders.....	87, 183	Hereditary cataract.....	25
subacute.....	129	retrobulbar optic neuritis.....	265
treatment of.....	87	treatment of.....	265
Glaucoma.....	19, 70, 194, 261	Herpes of the cornea.....	194
congenital.....	33, 34	of the lids.....	80
treatment of.....	34	treatment of.....	80
differential diagnosis.....	261	vesicles.....	80
infantile.....	33	Heterochromia.....	41
Glaucomatous halo.....	264	iris congenita.....	24
Glia coat, persistence of.....	32	Heterophoria.....	310
Glioma.....	19, 33, 109	Hordeoli.....	73
of the retina.....	281	Hordeolum.....	85, 94
Globe, considerably dislocated.....	271	externum.....	94
retraction of the.....	355	internum.....	70, 94
Globes, hypertrophy of.....	14	treatment of.....	94
Gonoblennorrhœa.....	154	Horner's symptom.....	308
Gonoblennorrhœal conjunctivitis, acute		Hutchinson's triad.....	215
infantile, treatment of.....	166		

	PAGE		PAGE
Hyaloid artery, persistence of the posterior part.....	37	Infantile eye, size of.....	52
persistent.....	19, 31	spasm of accommodation.....	62
Hydatoid cysts.....	189	sphincter pupillæ of.....	50
Hydrophthalmos.....	15, 19, 33	strabismus.....	54
congenitus.....	33	vitreous changes of.....	51
Hyperæmia, active, and inflammatory		weight of.....	52
swelling of one or both lids....	70	zonula of.....	51
and catarrh of the conjunctiva....	127	scurvy.....	69
of lids.....	70	Inflammation of the lachrymal glands,	
treatment of.....	70	treatment of.....	112
Hyperæmic chemosis.....	188	of the orbit.....	321
Hyperhidrosis.....	8	Inflammatory blepharospasm.....	101
Hyperkeratosis universalis.....	98	swelling of the tissue.....	125
treatment of.....	98	Influenza.....	129
Hypermetropia.....	4, 289	Injuries of the eye.....	331
absolute.....	289	of the orbit.....	353
facultative.....	289	Instillation of drops.....	132
treatment of.....	289	Insufficient general nutrition.....	191
Hyperopia.....	44	Interstratified form.....	148
Hypertonus.....	294	Inunction of lids.....	91
Hypertrichosis.....	8	Iridectomy.....	28
Hypertrophy of the globes.....	14	in infants.....	56
Hypopyon.....	206	Irideremia.....	16, 19, 30, 231
Hysterical ptosis.....	107	treatment of.....	30
		Iridochorioiditis, sympathetic.....	356
Ichthyosis congenita.....	98	Iridocyclitis.....	70, 206, 232, 236
Icterus.....	126	serous.....	247
Idiocy, Mongolian.....	8	syphilitic.....	236
Impetiginosa.....	136	Iridodialysis.....	28
Impetiginous eczema.....	79	Iridonesis.....	230
Incomplete paralytic ptosis.....	108	Iridoplegia.....	301
Indirect consensual reaction.....	306	coloboma of.....	14, 16, 30
Infantile eye, ability to fully appreciate		congenital absence of.....	19
space.....	60	displacement of.....	230
canal of the optic nerve.....	49	gumma of the.....	237
changes of orbital shape.....	49	in infants.....	50
color-sense of.....	60	serous cysts of.....	20
congenital myopia of.....	62	Iritis, papulous.....	237
cornea.....	50	Irritation and spasm of the external	
development of.....	47	ocular muscles.....	316
eyelids of.....	49		
fovea centralis.....	51	Keratitis.....	136, 204
hemorrhage of the retinal		annular.....	213
veins.....	53	bullous.....	196
iridectomy of.....	56	dendritica.....	195
iris of.....	50	disciformis.....	198, 350
optic disc.....	53	treatment of.....	198
nerve in.....	51	following lagophthalmos.....	205
ora serrata.....	51	interstitialis profunda.....	211
orbit of.....	49	local treatment of.....	206
pupil of.....	50, 57	neuroparalytica.....	206
pupillary reflex.....	58	prognosis of.....	206
refraction in new-born.....	61	pannosa.....	202
shape of.....	52	parenchymatous.....	262
		treatment of.....	216

	PAGE		PAGE
Keratitis, punctate.....	213	Lens, subluxation of.....	351
syphilitic.....	211	Lenticonus posterior.....	23
vesicular.....	196	treatment of.....	23
Keratoconus.....	14, 191, 224	Lenticular nucleus, displacement of....	23
treatment of.....	224	Leprosy.....	88
Keratoglobus.....	191	corneal changes in.....	223
Keratomalacia.....	203	treatment of.....	223
Koch-Weeks's bacillus, conjunctivitis		treatment of.....	88
of.....	135	uveal affections in.....	250
due to.....	128	treatment of.....	250
infection.....	149	Leprous affections of the conjunctiva..	184
Labyrinthal epiphora.....	114	Leucoma, congenital.....	14
Lachrymal abscess.....	11	Leucosis retinae.....	20
bone.....	120	Levator palpebrae, affections of.....	107
canals, blennorrhœa of.....	118	treatment of.....	108
treatment of.....	118	Lid, absence of Bell's phenomenon upon	
duct.....	3	closure of.....	43
congenital anomalies of the..	9	affections, prognosis of.....	75
strictures in.....	117	Lids, anthrax infection of.....	77
fistulae.....	9, 11	chancres of.....	83
gland.....	3, 110	circulatory disease of.....	69
glands, inflammation of.....	111	congenital anomalies of.....	5
diagnosis of.....	111	diagnosis of vaccine infection of..	75
tuberculosis of.....	112	eczema of.....	89
groove, persistent patency of....	9	prognosis of.....	90
organs, affections of.....	110	treatment of.....	90
passages, foreign bodies in.....	331	elephantiasis mollis of.....	33
sac.....	110	erysipelas of.....	76
acute dacryocystitis and blen-		differential diagnosis.....	76
norrhœa of.....	118	fissure, shortening of.....	5
congenital fistulae of.....	9	furuncles of.....	80
occlusion of.....	10	gangrene of.....	78
stenosis of.....	10	treatment of.....	79
differential diagnosis.....	10	habitual twitching of.....	99
strictures in.....	117	herpes of.....	80
suppuration of.....	10, 76	treatment of.....	80
secretion.....	110	hyperæmia of.....	70
Lachrymation, differential diagnosis of	115	treatment of.....	70
partial or total arrest of.....	113	inunction of.....	91
pathological.....	113	lower, cysts of.....	19
treatment of.....	115	neurofibroma of.....	6
treatment of.....	116	œdema of.....	71
Lagophthalmos.....	103	phlegmonous inflammations and	
Laminated cataract.....	24	abscesses of.....	79
Landolt's ophthalmodynamometer.....	310	treatment of.....	80
Lens, affections of.....	257	retraction and flattening of.....	67
diagnosis of.....	259	spontaneous hemorrhage and cir-	
prognosis of.....	259	culatory disease of.....	69
treatment of.....	260	tuberculosis of.....	81
coloboma of.....	27	local treatment of.....	81
congenital anomalies of.....	23	twitching of the upper.....	42
ectopia of.....	33	vaccine infection of.....	74
opacities of.....	30, 257	variola of.....	71
treatment of.....	30	Ligamentum pectinatum, fetal, persist-	
		ence of.....	14

	PAGE		PAGE
Lipodermoids.....	15	Muscles, orbicularis, spasm of.....	99
of the conjunctiva.....	12	Myasthenia.....	107
Lupus vulgaris.....	81	Myasthenic ptosis.....	107
Lymphangioma tuberosum multiplex..	109	Myopia.....	44, 290
Lymphatica.....	136	congenital.....	4
Lymph-cysts.....	189	in infants.....	62
Lymphoma of the conjunctiva.....	186	treatment of.....	294
treatment of.....	186	Myxosarcoma.....	109, 283
Macular coloboma.....	18	Nævi.....	6
disease.....	44	non-pigmented, of the conjunctiva	12
syphiloids.....	83	pilosi.....	6
Marantic necrosis of the cornea.....	203	Nasal accessory sinuses, affections of..	321
Marginal opacities.....	14	Nasolachrymal canal, osseous, tuber-	
Massage of the eye.....	221	culous caries of.....	120
Measles.....	129	duct.....	110
Mechanical ectropium.....	106	anomalies of.....	110
Medullated nerve-fibres.....	36	tuberculosis of.....	120
Megalocornea.....	33	Neoplasms of the orbit.....	329
Megalophthalmos.....	33	treatment of.....	330
Melanosarcoma.....	6	Nephritis, optic nerve in.....	279
Membrane, pupillary.....	8	Nerves, ciliary, neurofibroma of.....	33
Membranous atresia.....	10	facial, paralysis of.....	68
Meningitic neuritis.....	270	motor, paralysis of.....	41
Metastatic, abortive gonorrhœal con-		ocular, congenital disorders of....	41
junctivitis.....	168	oculomotor, spasm of.....	68
gangrene.....	78	optic, canal of, in infants.....	49
Microblepharia.....	8	coloboma of.....	37
Microcornea.....	14, 191	in infants.....	51
Microphthalmos.....	6, 8, 16, 18, 42	in nephritis.....	279
with orbitopalpebral cysts.....	39	in oxycephalia, changes in....	280
Mikulicz disease.....	112	peripheral neuritis of.....	269
Miliary phlyctenular conjunctivitis...	138	differential diagnosis.....	269
tuberculosis of the anterior uveal		treatment of.....	272
segment.....	244	simple atrophy of.....	266
Molluscum contagiosum.....	109	diagnosis of.....	267
Mongolian fold.....	7	treatment of.....	267
idiocy.....	8	tumors of.....	283
Mongolism.....	5	treatment of.....	283
Monolateral squinting.....	313	Nerve-fibres, medullated.....	36
Motility of the ocular muscles, con-		Neurasthenic weakness of accommoda-	
genital defects of.....	43	tion.....	302
Motor nerves, paralysis of.....	41	Neuritis, meningitic.....	270
Mucocele.....	322	optic.....	44, 270
Mucosa, tuberculous ulcers of.....	120	syphilitic.....	270
treatment of.....	120	tubercular optic.....	270
Muscles, congenital disorders of.....	41	Neurofibroma of the ciliary nerves....	33
external, of the eye, affections of..	310	of the facial skin and orbit.....	33
ocular, external, irritation and		of the lids.....	6
spasm of.....	316, 317	Neurotic facial atrophy.....	98
paralysis of.....	317	treatment of.....	99
diagnosis of.....	318	New-born, acute pemphigus of.....	97
etiology of.....	317	refraction of the eyes in.....	61
symptoms of.....	318	weeping in.....	62
traumatic paralysis of.....	353	New growths of the conjunctiva.....	189

	PAGE		PAGE
Night-blindness.....	4, 44	Optic nerve, peripheral neuritis of,	
Non-pigmented nævi of the conjunctiva	12	treatment of	272
Non-specific blennorrhœa neonatorum	165	simple atrophy of.....	266
Normal conjunctiva.....	125	diagnosis of	267
Nutrition, general, insufficient.....	191	treatment of	267
Nystagmus.....	4, 8, 19, 33, 40, 44	tumors of.....	283
		treatment of	283
Ocular affections, examination of, in		neuritis.....	44, 270
children.....	63	vesicle.....	1
anomalies due to ancestry, heredity,		Ora serrata, in infants.....	51
or consanguinity.....	44	Orbicularis muscle, spasm of.....	99
muscles, external, irritation and		Orbit, congenital anomalies of.....	38
spasm of.....	317	dermoid cysts of.....	38
treatment of	317	diseases of.....	321
paralysis of	317	of infantile eye	49
diagnosis of	318	inflammations of.....	321
etiology of	317	differential diagnosis.....	322
symptoms of	318	prognosis of	322
traumatic paralysis of	353	treatment of	323
nerves, congenital disorders of....	41	injuries of.....	353
Oculomotor nerve, spasm of.....	68	prognosis of	354
Œdema of the lids.....	71	treatment of	355
Oligidrosis.....	8	neoplasms of.....	329
Opacities, congenital, of the cornea....	14	treatment of	330
of the cornea.....	6	serous infiltration and phlegmon of	325
of the lens.....	30	differential diagnosis	325
marginal.....	14	treatment of	326
Ophthalmia due to caterpillar hairs	336	tuberculosis of.....	327
treatment of	339	treatment of.....	329
gonorrhœal	153	Orbita, neurofibroma of.....	33
pseudo-tuberculosis	336	Orbital cysts	19
treatment of	339	congenital serous.....	40
sympathetic.....	356	hemorrhage.....	125, 329
prognosis of	357	treatment of	329
treatment of	357	inflammations.....	321
Ophthalmoblennorrhœa.....	155	periostitis.....	321, 326
course of.....	156	treatment of	327
Ophthalmodiaphanoscope.....	20	Orbitopalpebral cysts, microphthalmos	
Ophthalmodiaphanoscopy.....	18	and anophthalmos with.....	39
Ophthalmogonorrhœa, acute.....	155		
Optic cup.....	1	Palpebral abscess.....	76
disc.....	263	coloboma.....	4
in infants.....	53	fissures, gaping of.....	69
grooves.....	1	Pannus of the cornea.....	171, 202
muscles, paresis of	8	Panophthalmitis.....	225, 252
nerve, affections of.....	263	differential diagnosis in.....	158
aplasia of.....	38	Papilloma of the conjunctiva.....	189
canal of, in infants.....	49	treatment of	190
coloboma of.....	37	Papular syphiloids.....	83
congenital anomalies of.....	35	Papulous iritis.....	237
in infants.....	51	Paralysis of accommodation.....	301, 302
in nephritis	279	as a late manifestation of	
in oxycephalia, changes in	280	syphilis.....	303
peripheral neuritis of.....	269	of the facial nerve.....	68
differential diagnosis	269	of the motor nerves.....	41

INDEX

491

	PAGE		PAGE
Paralysis of the ocular muscles.....	317	Polyps.....	89
etiology of.....	317	Polysyndactylism	19
of the trigeminus	104	Posterior, lenticonus	23
Paralytic ptosis	107	polar cataract.....	23, 25
incomplete	108	synechia, congenital.....	20
Paralytical strabismus.....	4	Prophylaxis.....	149, 175
Parenchymatous keratitis	211, 262	Pruriginosus foliaceus	97
differential diagnosis	214	Pseudoglioma	282
treatment of	216	Pseudoneuritis	36
xerosis	186	Pseudopterygium	187
Partial absence of the iris, congenital..	19	treatment of	187
or total arrest of lachrymation....	113	Pseudo-ptosis.....	107
Pathological lachrymation.....	113	Pseudo-strabismus	314
treatment of	115	Pterygoid.....	187
Pemphigus	149, 152, 183	Ptosis	8, 41
diagnosis of	152	adipose.....	43
prognosis of	153	atonic.....	43
treatment of	153	bilateral.....	5
Perforations of the palpebral skin.....	344	differential diagnosis of	107
Pericystitis lacrimalis.....	118	hysterical.....	107
Periostitis, orbital	321, 326	myasthenic.....	107
syphilitic.....	327	paralytic	107
treatment of	327	incomplete.....	108
treatment of	327	sympathetic	108
Peripheral neuritis of the optic nerve	269	treatment of	42
Persistence of the fetal ligamentum		Pulsating exophthalmos.....	355
pectinatum.....	14	Puncta lacrimalia, atresia of.....	9
Persistent hyaloid artery	19	Punctate keratitis.....	213
patency of the lachrymal groove..	9	Pupillary function, examination of ...	305
Persisting remains of the tunica vascu-		membrane.....	8
losa lentis	27	movements, disturbances of, treat-	
Pertussis.....	69	ment of	300
Petechiae	69	reaction in the infectious dis-	
Phlebotomy	228	eases.....	309
Phlegmonous dacryocystitis.....	10	reflex in infants.....	58
inflammations and abscesses of the		rigidity in hysterical and epileptic	
lids	79	paroxysms	306
Photophobia.....	40, 131, 144	Pupils, congenital inequality of	306
Phthiriasis palpebralis.....	93	in infants.....	50, 57, 305
pubis	93	unequal.....	306
Phthisis bulbi	206	Pustular conjunctivitis.....	138
Phlyctenulosa maligna	138	Pustulous eczema.....	93
Phlyctenuloso-pustulosa.....	136	Pyocyanase	210
Phlyctenular conjunctivitis, prognosis			
of	139	Recurring vitreous hemorrhage	251
Phlyctenules	136	Reflex deafness.....	307
Pigment degeneration of the retina	265	pupillary rigidity.....	307
Pigmentation, congenital anomalies of	40	spasms	99
Plague conjunctivitis.....	129	Refraction, anomalies of.....	285
Pneumococcus conjunctivitis.....	149	Relative range of accommodation....	301
Polar cataract, anterior.....	25	Reticular injection	125
posterior	23, 25	Retina, affections of	263
Poliosis, circumscribed.....	40	aplasia of	38
Polycoria.....	21	congenital anomalies of.....	35
Polymyositis	107	cyanosis of	36

	PAGE		PAGE
Retina, detachment of.....	294	Septic suppurative keratitis, treatment	
glioma of.....	281	of.....	208
diagnosis of.....	282	Sequelæ.....	172
prognosis of.....	282	Serous chemosis.....	188
treatment of.....	282	cysts of the iris.....	20
pigment degeneration of.....	265	epithelial cysts.....	189
treatment of.....	266	infiltration and phlegmon of orbit.....	325
Retinal veins, hemorrhage of in infants	53	iridocyclitis.....	247
vessels.....	2	Serpiginous ulcer of cornea.....	207
Retinitis, albuminuric.....	275	Shape of infants' eyes.....	52
pigmentosa.....	4, 44, 46, 265	Signs of degeneration.....	5
punctata albescent.....	19	Sinuses, nasal accessory, affections of..	321
Retraction of the globe.....	355	Size of infants' eyes.....	52
of the lids.....	67	Skin, tuberculous ulcer of.....	81
Roseola.....	83	Smallpox.....	129
Sac, cystic dilatation of.....	114	Soft chancre.....	86
lachrymal.....	110	Space, ability to fully appreciate, in	
acute dacryocystitis and blen-		infants.....	60
norrhœa of.....	118	Spasm, accommodative.....	294
congenital fistulæ of.....	9	of the oculomotor nerve.....	68
strictures in.....	117	of the orbicularis muscle.....	99
Salvarsan.....	217, 243	reflex.....	99
Sarcoma, soft.....	109	Spasmus nictans.....	99
Sarcomatosum.....	106	Sphincter pupillæ, in infants.....	50
Schlemm, venous spaces of.....	34	Spring catarrh.....	174, 184
Schlemm's canal.....	15	differential diagnosis.....	185
plexus.....	14	treatment of.....	185
absence of.....	14	Squamous blepharitis.....	89
Sclera, affections of.....	225	Squint, concomitant.....	311
differential diagnosis.....	226	treatment of.....	315
treatment of.....	226	upward.....	314
circumscribed pathological pig-		Staphyloma, congenital.....	15
ment spots of.....	41	of the cornea.....	14
congenital change of.....	22	Stellwag's symptom.....	69
thinning of.....	22	Stenosis of lachrymal sac.....	10
treatment of.....	22	Strabismus.....	4, 44
Scleral cysts, congenital.....	22	concomitans.....	311
translumination.....	20	concomitant.....	4
Scleritis.....	226	convergent, Worth treatment in..	316
Scleroderma.....	98	difference between concomitant	
Scotoma, central.....	270	and paralytic.....	312
Scrofula, diet in.....	141	in infants.....	54
feeding in.....	141	paralytical.....	4
Scrofuloderma.....	81	vertical.....	314
treatment of.....	82	Streptothrix actinomycosis.....	115
Scrofulosis.....	129	Strictures in the lachrymal sac and duct	117
Scrofulous ophthalmia.....	136	Subacute glanders.....	129
processes of the cornea.....	202	Subconjunctival hemorrhages.....	187
Scurvy, infantile.....	69	serous retention cysts.....	189
Semilunar fold.....	13	Subluxation of the lens.....	351
Septic stab wound.....	354	Suppuration of the lachrymal sac....	10, 76
suppurative keratitis.....	207	Swelling of the tissue.....	125
differential diagnosis.....	208	Sycosis.....	89
prognosis of.....	208	Symblepharon.....	6, 8
		operation.....	346

	PAGE		PAGE
Sympathetic ophthalmia.....	356	Trichiasis.....	88, 105
ptosis.....	108	and distichiasis.....	97
Symptomatic weakness of accommo- dation.....	302	partial.....	97
Synechiæ, congenital anterior and pos- terior.....	20	treatment of.....	98
Syphilides, gummatous.....	84	Trichopytia profunda.....	92
Syphilis.....	274	Trigeminus, paralysis of.....	104
gummatous syphilitic tarsitis in hereditary or acquired.....	85	Tubercular optic neuritis.....	270
Syphilitic affections.....	82, 236	Tuberculosis, bovine conjunctival.....	183
of the conjunctiva.....	179	colliquata.....	81
diagnosis of.....	182	treatment of.....	82
differential diagnosis.....	179	of the conjunctiva.....	182
eruptive forms.....	83	differential diagnosis of.....	183
treatment of.....	84	treatment of.....	183, 184
of the nasolachrymal canal and sac.....	120	of the cornea.....	215
treatment of.....	83	of the lachrymal glands.....	112
iridocyclitis.....	236	of the lids.....	81
keratitis.....	211	local treatment.....	81
keratomalacia.....	204	luposa.....	81
neuritis.....	270	of the nasolachrymal duct.....	120
orbital periostitis.....	327	of the orbit.....	327
treatment of.....	327	treatment of.....	329
tarsitis, acute.....	86	ulcerosa miliaris.....	81
treatment of.....	86	uveal.....	244
ulcers.....	183	diagnosis.....	245
Syphiloids, macular and papular.....	83	differential diagnosis.....	244
		prognosis.....	246
		treatment of.....	246
Test-boards.....	286	Tuberculous caries of the osseous naso- lachrymal canal, and bone.....	120
Tests, functional.....	63	ulcers of the mucosa.....	120
Tetanus, traumatic.....	354	treatment of.....	120
Thinning of the sclera.....	22	of the skin.....	81
Tissue, inflammatory swelling of.....	125	local treatment of.....	81
Tortuositas vasorum.....	36	Tumors.....	109
Total absence of the iris, congenital....	19	of the optic nerve.....	283
Toxic amblyopia.....	274	treatment of.....	109
Trabeculæ, fetal.....	20	uveal.....	256
Trachoma.....	169	Tunica vasculosa.....	3
diagnosis of.....	173	lentis.....	3
prognosis of.....	174	persisting remains of.....	27
treatment of.....	174	Twitching of the lids.....	99
Transitory elevation of the blood- pressure.....	126	of the upper lid.....	42
Translumination, scleral.....	20	Typhus fever.....	129
Transplantation of muscle in congenital defects of motility of ocular muscles	43	Ulcerating blepharitis.....	91
Traumatic cysts.....	20, 189	Ulcers, annular.....	200
paralyses of the ocular muscle....	353	treatment of.....	201
tetanus.....	354	exzematous, of the cornea.....	198
Treatment, local, of the seborrhœa of the palpebral margins.....	93	syphilitic.....	183
of parenchymatous keratitis.....	216	tuberculous, of the mucosa.....	120
of tumors.....	109	Ulcus molle.....	86
		Unilateral blennorrhœiform conjuncti- vitis.....	130
		metastatic suppurative uveitis....	252
		Upward squint.....	314

	PAGE		PAGE
Uveal affections in leprosy	250	Verruca plana juvenilis	109
coloboma	6	Vesicle, germinal	1
irritation and inflammation	351	herpes	80
tract, congenital anomalies of	16	optic	1
diseases of	230	Vesicular keratitis	196
differential diagnosis	234	Vessels, cilioretinal	35
prognosis of	234	retinal	2
treatment of	234	Vision tests	287
syphilitic affections, prognosis		Visual disturbances	263
of	242	in childhood	298
treatment of	243	from diseased upper teeth	271
tuberculosis	244	due to loss of blood	268
diagnosis	245	treatment of	268
differential diagnosis	244	Vitreous body	2
treatment of	246	changes in infants	51
tumors	256	congenital anomalies of	31
		opacity and shrinking	340
Vaccine blepharitis	74	Warts	109
infection of the lids	74	Weil's disease	129
diagnosis of	75	Weight of infants' eyes	52
Vaccinola, treatment of	146	Worth treatment in convergent stra-	
and varicellæ	146	bismus	316
Varicellæ	146	Xeroderma pigmentosum	98
treatment of	146	Xerophthalmos	173
Variola, differential diagnosis	72	Xerosis, conjunctival	203
of the lids	71	of cornea	104
treatment of	72	Zonula	2
Variolar pustules of the conjunctiva ..	145	in infants	51
Vasa hyaloidea propria	3		
Veins, retinal, hemorrhage of, in infants	53		
Venous spaces of Schlemm	34		

PART II

DISORDERS OF SPEECH AND PHONATION *

	PAGE		PAGE
Absence of orientation.....	394	Blindness.....	378
Accentuation.....	379	word, congenital.....	476
Acoustic attention.....	381, 392	Bradyllalia, spastic.....	458
"Acoustic reflex".....	359	Bridgemann, Laura.....	384
Acquired sound-language.....	379	Bulbar disorders.....	454
Agrammatism.....	394	paralysis.....	454
causes and origin.....	394		
diagnosis of.....	395	Causes and origin of auditory dumb-	
prognosis of.....	395	ness.....	388
symptoms.....	395	Cerebral diplegia.....	457
treatment of.....	396	complicated by bilateral athe-	
Akataphasia.....	382, 387, 394	tosis or chorea.....	457
causes and origin.....	394	paralysis, disorders of speech due	
diagnosis of.....	395	to.....	456
of the imbecile.....	449	syphilis.....	454
prognosis of.....	395	Children partly or totally deaf, treat-	
symptoms.....	395	ment of.....	382
treatment of.....	396	Chorea minor, disorders of speech in..	436
Aphasia.....	460	Choreic aphasia.....	462
after injury.....	461	Chronic hoarseness.....	466
apoplectic.....	462	Comprehension, lingual.....	373
and bulbar disorders.....	454	Congenital disorders of speech.....	440
in childhood.....	459	word-blindness.....	476
choreic.....	462	Contaminations.....	373
hysterical.....	438, 460	Cry-cure.....	360
in infantile paralysis.....	461		
motor.....	462	Deaf-blindness.....	384
in other organic affections.....	461	congenital.....	384
reflex.....	460	Deaf-mutes.....	378
sensory.....	387, 462	Deafness, monotony of the voice in...	382
voluntaria.....	462	lingual.....	387
Aphonia, hysterical.....	439	total or partial.....	378
Aphthongia.....	417	Deaf persons, instruction in.....	386
Apoplectic aphasia.....	462	Dementia infantilis.....	443
Articulating exercises.....	474	Dental sounds, stammering in.....	397
Articulation, exercises in.....	431	Dentition, process of.....	363
various spasms.....	419	Dento-orthopædic correction.....	403
Associative-reproductive.....	368	Development, differences in.....	375
Ataxia, hereditary.....	451	general physico-psychic.....	361
Attention, acoustic.....	392	idiomatic.....	361
motor.....	392	lingual arrests of.....	378
Audimutitas.....	388, 460	peripheral expressive arrests of	399
Auditory dumbness.....	388	preliminary stages of.....	361
causes and origin of.....	388	of sentences.....	374
diagnosis of.....	389	of speech.....	359
differential diagnosis.....	389	Differences in development.....	375
examination in.....	389	Disorders of speech.....	412
		due to affections of the peri-	
"Baby-talk".....	363, 367	pheral nervous system and	
Balbuties.....	412	the muscular apparatus....	459

* For index of disorders of the eye, see page 481.

	PAGE		PAGE
Disorders of speech due to encephalitis	455	Formation of sentences	373
due to infantile cerebral para-		Functional stammering	396, 397
lysis	456		
due to paralysis of the facial		Gammacism	397
nerves	459	Gesture language	379
due to sclerosis of the central		Gestures	378
nervous system	455	Gutzmann's clinical division of stut-	
in chorea minor	436	terers	416
in epilepsy	436	method	429
in functional diseases of the		Hearing, impaired, speech in	380
nervous system	436	Hereditary ataxia	451
in hysteria	438	Hoarseness, chronic	466
in maladie des tics	437	Hysteria, disorders of speech in	438
owing to organic affections of		Hysterical aphasia	438, 460
the nervous system	451	aphonia	439
in partially deaf imbeciles	446	mutism	438
at puberty	434	stuttering	438
of the voice	463		
Disturbance of lingual accents in the		Idiomatic development	361
imbecile	450	Imbecile, akataphasia of the	449
treatment of	450	disorders of speech in partially	
of speech in imbeciles	440	deaf	446
of the voice	466	disturbances of speech in	440
of the voice at puberty	467	lingual accents in, disturbance of	450
Dumbness, auditory	388	treatment of	450
course of	390	mutism in	441
diagnosis of	389	diagnosis of	443
differential diagnosis	389	treatment of	445
examination in	389	stammering of	448
prognosis of	390	treatment of	448
treatment of	390	stuttering of the	449
Duplicating syllables	373	Imitation of sounds	362
Dysacusis	382	intentional	363
Dysarthric disturbances	439	Importance and prophylaxis of dis-	
Dyslalia	396	orders of speech and voice in school	469
causes and origin of	396	Inability to learn to read	476
labialis	411	to read	477
mechanical	399	etiology of	477
nasal	405	treatment of	478
Echolalia	363, 371, 437, 446	Infantile pseudobulbar paralysis	458
Emotional-volitional	367	voice	463
Encephalitis, disorders of speech due to	455	persistence of	467
Enunciation, mistakes of	372	Infants, deaf, congenitally	378
Epilepsy, disorders of speech in	436	Instruction in deaf persons	386
Exercises, articulating	474	Intellectual understanding	367
reading	474	Irritation with coughing and interrup-	
respiratory	473	tion of speech	440
External development of words	371		
		Keller, Helen	384
Facial nerves, disorders of speech due			
to paralysis of	459	Labial sounds, stammering in	397
paralysis	459	Lallation, spontaneous	361
spasms	437	Language, complete, physiology of	376
First independent talking	366	gesture	379
		Larynx, movements of the	418
		Letters, omission of	373

INDEX

497

	PAGE		PAGE
Lingual arrests of development.....	378	Parasigmatism.....	402, 459
comprehension.....	373	Partial deafness.....	378
deafness.....	387	Peripheral expressive arrests of lingual	
defects.....	383	development.....	399
Lips, spasms of.....	419	Peripherally impressive arrests.....	378
Lisping.....	400	Persistence of the infantile voice....	467
causes and origin.....	400	Persistent falsetto voice.....	467
diagnosis of.....	402	Perverse imitation in girls.....	467
nasal.....	404	Physico-psychic development.....	361
prognosis of.....	402	Physiologica.....	460
treatment of.....	403	Physiological stammering.....	372
Logical lingual intelligence.....	369	Physiology of the complete language..	376
L-stammering.....	398	of the development of speech.....	359
Macroglossia.....	401	of the infantile voice.....	463
Mechanical dyslalasia.....	399	"Plural conception".....	370
Metathesis.....	373	Polio-encephalitis, acute.....	455
Microglossia.....	401	Polyneuritis.....	459
Mistakes of enunciation.....	372	Preliminary stages of lingual develop-	
Modulation.....	377	ment.....	361
Monotony of the voice in deafness....	382	Primitive sounds.....	362
Motor aphasia.....	462	Prolonged mutation.....	467
attention.....	392	Prophylaxis of the lingual and vocal	
Motor-kinesthetic.....	360	disorders in school.....	472
Multiple sclerosis.....	455	Psychology and physiology of the de-	
Mutation, perverse, in girls.....	467	velopment of speech.....	359
prolonged.....	467	Psychotherapy.....	428
of the voice.....	465	Puberty disorders of speech at.....	434
Mutism in childhood.....	459	disturbances of the voice at.....	467
hysterical.....	438	treatment of.....	468
in imbeciles.....	441	stuttering at the time of.....	434
diagnosis of.....	443	Reading exercises.....	474
treatment of.....	445	Reflex aphasia.....	460
Mutismus hystericus.....	460	"Relative stage".....	371
Mutitas.....	460	Respiration.....	417
idiotica.....	441, 460	Respiratory exercises.....	429, 473
physiologica.....	459	Rhinolalia.....	405
"Nasal Parasigmatism".....	402	aperta.....	406
Nerves, facial, disorders of speech due		functionalis.....	408
to paralysis of.....	459	organica.....	407
Nodules, singers'.....	466	causes and origin.....	405
Omission of letters.....	373	clausa.....	406
"One-word sentence".....	366	anterior.....	406
"Oral stuttering".....	415	functionalis.....	406
Orientation, absence of.....	394	posterior.....	406
Palatal sounds, stammering in.....	397	mixta.....	406
Paralysis, bulbar.....	454	anterior.....	408
facial.....	459	posterior.....	408
of the facial nerves, disorders of		R-stammering.....	398
speech due to.....	459	treatment of.....	398
infantile, aphasia in.....	461	School, importance and prophylaxis of	
pseudobulbar.....	458	disorders of speech and voice in.	469
spastic family.....	451	singing in.....	475
Paraphrasia præceps.....	392		

	PAGE		PAGE
Schools, stuttering in, treatment of		Speech, dysarthritic	476
cured or improved	471	in impaired hearing	380
Schulz, Hertha	384	lipping	476
Sclerosis of the central nervous system,		psychology and physiology of the	
disorders of speech due to	455	development of	359
multiple	455	Spluttering	387, 392
Sensory aphasia	387, 462	diagnosis of	393
Sentences, development of	374	prognosis of	393
formation of	373	treatment of	393
Sigmatism	400	Spontaneous lallation	361
addental	401	"Stage of lingual substance"	370
interdental	401	Stammering	387
labio-dental	401	in dental sounds	397
lateral	402	differential diagnosis	399, 425
nasal	402	functional	396, 397
strident	401	causes and origin	396
treatment of	404	treatment of	397
Singers' nodules	466	of imbeciles	448
Singing in school	475	treatment of	448
Snuffling	405	in labial sounds	397
diagnosis of	408, 409	organic	399
prognosis of	409	in palatal sounds	397
treatment of	410	physiological	372
Sound-language, acquired	379	prognosis	399
Spasms of articulation	419	syllable and word	398
facial	437	diagnosis of	398
of the lips	419	treatment of	399
Spastic bradylalia	458	vowel	397
family paralysis	451	treatment of	397
Speech, development of	359	Stutterers, Gutzmann's clinical divi-	
acoustic-optical	359	sion of	416
general physico-mental	359	Stuttering	412
motor-kinæsthetic	359	causes and origin of	413
retarded	476	congenital taint	414
disorders of	412	diagnosis of	424
in chorea minor	436	differential diagnosis	425
congenital and early acquired	440	etiology of	426
due to affections of the peri-		hereditary taint	414
pheral nervous system and		hysterical	425, 438
the muscular apparatus	459	prognosis of	439
due to encephalitis	455	treatment of	439
due to infantile cerebral paral-		of the imbecile	449
ysis	456	occurrence of	413
due to sclerosis of the central		oral	415
nervous system	455	periodical	415
in epilepsy	436	prognosis of	425
in functional diseases of the		prophylaxis in	432
nervous system	436	at the time of puberty	434
in hysteria	438	diagnosis of	435
in maladie des ties	437	prognosis of	435
owing to organic affections of		symptomatology	434
the nervous system	451	treatment of	435
in partially deaf imbeciles	446	in schools, treatment of cured or	
at puberty	434	improved	471
disturbances of	476	treatment of	426, 431

INDEX

499

	PAGE		PAGE
Stuttering, symptomatology	417	Voice, exercising the.....	474
Syllable and word-stammering.....	398	falsetto, persistent.....	467
Syllables, duplicating.....	373	infantile.....	463
Symptomatology.....	392, 406	persistence of.....	467
of congenital word-blindness.....	476	physiology of.....	463
Syphilis, cerebral.....	454	monotony of, in deafness.....	382
Talking, first independent.....	366	mutation of.....	465
Total or partial deafness, blindness....	378	of the nursing.....	463
Treatment of children partly or totally		Vowel-stammering.....	397
deaf.....	382	treatment of.....	397
Tumultus sermonis.....	392	Whispering.....	377
Tumefaction.....	454	voluntary.....	391
Verbigeration.....	437	causes and origin.....	391
Visual perceptions.....	359	Word-blindness, congenital.....	476
Voice, disturbances of.....	466	etiology of.....	477
at puberty.....	467	symptomatology of.....	476
treatment of.....	468	treatment of.....	478
disorders of.....	463	Word-stammering.....	398
		Words, external development of.....	371

1

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

~~JUN 27 1922~~

JUN 15 1939

NOV 17 1941

NOV 16 1973

Q48 Tversbusch, O. 46318
E93h Diseases of the eye in
1914 childhood, tr. by Howe &
Francis - Disorders of
speech, by Nadoleczny

L. J. J. J. J.
H. J. J. J. J.
L. J. J. J. J.
R. J. J. J. J.
J. J. J. J. J.

JUN 27 1922
JUN 15 1935
NOV 18 1941

